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THE SENSORY FUNCTIONS ATTRIBUTED TO THE SEVENTH NERVE*

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It is first necessary to define what is meant by the seventh or facial nerve as considered in this paper. If by it is meant that efferent trunk which arises from one, or as some hold two, nuclei, first taking its tortuous course through the substance of the pons to its superficial origin, thence through the internal auditory meatus and Fallopian aqueduct to the stylomastoid foramen, to be distributed to various portions of the face, the question would be divested of interest, as the nerve would at once be recognized by all as purely motor. Two other ways of looking at the seventh nerve must however be taken into consideration.

In the first place if the views of Head and his collaborators are accepted the question of an afferent system of fibers traveling in the periphery with the motor facial needs to be discussed. Head, it will be remembered, holds that three different systems of afferent fibers are concerned with peripheral sensibility. Two of these, called respectively the epicritic and protopathic systems, are cutaneous. In consequence of the possession of epicritic sensibility the individual is able to localize light touch, to recognize warmth and coolness, and to discriminate as to one or two points when two are applied. Protopathic sensibility endows him with the power of recognizing extreme degrees of heat and cold. Through the third system, called that of deep sensibility, comes a recognition of deep pressure, which may be painful, and of position and passive movement. Epicritic and protopathic sensibility are conveyed to the spinal cord through a system of afferent fibers which run from the skin; deep sensibility by fibers which run with the motor nerves in the muscles and probably have their peripheral end organs located in the tendons and joints (including perhaps the periosteum and bone). Applying these views to the seventh nerve, it follows that the afferent system concerned with deep sensibility, including the pressure and postural senses, might be conveyed brainward in the trunk of the facial, leaving it probably somewhere before it enters the cerebrospinal axis.

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In the second place the neural system composed of the geniculate ganglion and its distal and proximal outgrowth is commonly regarded on the basis of the homologies between spinal and cranial ganglia as the sensory portion of the seventh nerve. At the same time all are not agreed as to exactly what are the sensory functions of this afferent system of the seventh.

The questions which present themselves in detail are: (1) Do afferent fibers concerned with deep sensibility (deep pressure and the sense of position and of passive movement) run in the branches and trunk of the motor facial; (2) has cutaneous sensibility, epicritic and protopathic, any general representation in the distribution of the facial; (3) do afferent tracts concerned with common sensibility (epicritic and protopathic) proceed from limited areas in the throat, tongue and ear by way of the geniculate system of the facial to their sensory termini in the oblongata; (4) to what extent are the geniculate ganglion, the chorda tympani, and the great superficial petrosal nerves and the intermediary nerve of Wrisberg to be regarded as concerned in the function of taste?[1]

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[1] The bibliography appended to the article is not to be regarded as exhaustive, although consultation of the papers enumerated will enable the reader to trace the entire literature of the subject. I have given considerable time to a study of the embryological aspects of the subject, the work of Dixon and of Van Gehuchten having been found especially valuable in this connection.

In the preparation of this paper I have studied the conditions as to sensation in seven cases of section of the root or of the branches of the fifth nerve or of partial or complete extirpation of the gasserian ganglion. Great care was taken in examining these cases first to define accurately the limits of complete and of partial anesthesia, and thus to decide what

PRESSURE SENSE

As already indicated it would seem probable that if deep sensibility is to be relegated to a separate afferent system, the fibers of this system should run with the motor fibers proper, although in a different direction. This would bring the facial, and also the motor trigeminal in harmony with what Head believes to be the case in spinal systems.

Before taking up the pressure sense in connection with the discussion of the functions of the seventh nerve it might be well to say a few words about the manner in which pressure is represented by peripheral end organs. All investigators of this subject are not agreed that the sense of pressure is represented in parts beneath the skin. Some hold that it is in part so represented and in part may be cutaneous. Trotter and Davies, for instance, believe that it is a distinct form of sensation, but in some of its degrees at least, may be recognized by the skin, especially by the deeper cutaneous layers. Head and his collaborators bring light pressure into the same series with light or superficial touch, but they refer pressure sufficient to produce painful response to deep sensibility. Trotter and Davies believe that tactile discrimination, that is, the recognition of two points by the Weber compass test, is dependent upon the sense of pressure, rather than upon the epicritic sensibility of Head. The way of looking at this matter is of some importance in deciding the question of the representation of the pressure sense in the seventh nerve. If light or moderately hard pressure is recognized through the intermediation of cutaneous end organs,

branch or branches of the nerve were fully separated from their centers. The conclusions reached were in all cases based upon the study of cases in which the areas of distribution of either the second or the third or both branches were completely anesthetic.

For the opportunity of investigating these cases I am indebted to several of my colleagues, Drs. Charles H. Frazier, J. C. DaCosta and F. X. Dercum, of Philadelphia, and Dr. Harvey Cushing, of Baltimore. These gentlemen not only placed the cases at my disposal, but gave me valuable assistance in their study. Valuable assistance in examining these patients was given by Dr. Frederick Prime, of the surgical, and Dr. Edward Mercur Williams, of the neurological, staff of the University of Pennsylvania.

In addition to these cases, of trigeminal operation, to obtain data for the paper, I have studied ten cases of peripheral facial paralysis. For the opportunity of studying one of these cases I am indebted to Dr. William G. Spiller; for another to Dr. Charles S. Potts. The rest were observed in private practice or in my services at the hospital of the University of Pennsylvania and the Philadelphia General Hospital.

even if these are situated in the deeper layers of the skin, it is possible that this sense of pressure is represented not in the seventh, but in the fifth nerve, as the latter, as is well known, is the great sensory nerve to the skin, although by it other tissues are also sensorially innervated.

Dana, after division of the third branch of the fifth nerve, found absolute loss of all forms of sensation, including that of deep pressure, in all the parts supplied by this nerve. No anesthesia of the ear or meatus was present. The necessary conclusion on these findings was that sensation, including pressure sensibility, was not conveyed in those branches of the seventh nerve which are distributed to the muscles of the face generally. If conveyed at all by them it was only to the parts of the ear which were not anesthetic. This is of course presuming that the auricle and meatus are not supplied by cervical and other nerves than the fifth or seventh nerves. Dana has also reported a case of complete paralysis of the facial nerve, in which no loss of deep sensation or sense of position could be demonstrated.

Dana carefully describes the methods and precautions used by him in examining for pressure sensibility. He kept one finger inside of the mouth and the other outside, testing for pressure in this way with negative results. When, however, the pressure was exerted so as to affect the bone, the patient appreciated that something was being done to him in that particular place, as he did also when the anesthetic parts were stretched, this result evidently being attributed to the effect produced on neighboring sensitive parts.

Two cases were studied by Ivy and Johnson, both observed in the service of Spiller. In the first of these cases the right fifth nerve was destroyed by a tumor which also involved other parts, but not the seventh nerve. Ivy and Johnson found in this case that sensation of deep pressure was preserved over the fifth nerve areas, as pressure was always recognized by the patient with his eyes closed. In the other case the sensory root of the fifth nerve was cut, the section being complete as demonstrated by subsequent necropsy. In this case the sense of pressure was retained over the area of distribution of the fifth nerve, while that of touch was lost.

Spiller is cited as having observed a third case in which pressure sensibility was retained after destruction of the gasserian ganglion.

The conclusions of Ivy and Johnson were that the fifth nerve conveys fibers of sensibility to light touch, to changes in temperature, and to pain from those parts generally recognized as being within the area of distribution of the said nerve; that the seventh nerve conveys fibers of deep or pressure sensibility from the muscles which it supplies with motor fibers; and that the muscles of mastication, which derive their motor supply from the fifth nerve, probably are also furnished with fibers of deep sensation by this nerve.

In discussing their cases Ivy and Johnson refer to the work of Head, and also to that of Van Gehuchten, Spiller, Donath, Dana, Hunt, von Lenhossek, Retzius and Amabolino, to some of whom references will be made in other connections in the present paper.

In all the cases of trigeminal section investigated by me the pressure sense was studied with results which were practically uniform. In the first place I found it was essential that the condition as to cutaneous sensibility of the so-called epicritic and protopathic types should be very carefully determined. In some of the cases examined for instance, the impairment of sensation in one or more branches of the trigeminus was incomplete. This was the fact even in some cases in which the gasserian ganglion was supposed to have been extirpated, or the sensory root of the fifth cut or torn out. In only one case was the loss of cutaneous sensation in the three branches of the fifth nerve absolute. In several cases sensation was completely abolished in the distribution of the second branch. My conclusions were drawn only from the investigation of completely anesthetic parts.

The methods adopted for testing the sense of pressure were various, as by pressure with the finger, with a pencil, or some other hard object, the use of the ophthalmotonometer and the algometers of Cattell and of Head and Rivers. In two cases in addition the method was adopted of applying steadily increasing weight. With the patient lying down a small card or metal receptacle was placed in the anesthetic area of the cheek and then weights were gradually added, beginning with less than a grain and increasing until a half ounce or more was reached. During all the pressure examinations the patient was blindfolded or his eyes were closed.

The results obtained by these examinations were practically uniform and indicated that the sense of pressure was represented in the fifth and not in the seventh nerve. In one case when severe pressure was exerted with the algometer over the upper maxilla the patient stated that he thought he felt a point of some kind, but seemed somewhat uncertain about it. The same individual in other examinations could not recognize anything. Whenever the pressure was exerted between the fingers or with one finger in the mouth, using a pressing object on the outside no response could be obtained.

In all the cases of seventh nerve paralysis examined no alteration in the sense of pressure could be determined, although in three cases the patients stated that they felt all forms of stimulus including pressure less upon the paralyzed than upon the other side. These observations I shall presently discuss. The results were probably due to suggestion.

Some of Cushing's observations on the tongue might be interpreted as indicating the presence of a pressure sense in a part supplied by the resected trigeminus, but the conclusion seems doubtful. By the ordinary methods of examination by a hair, pin, compass points, etc., no sensation was evoked, but the patient recognized a swab or some similar object drawn over the tongue. The tongue is an exceedingly mobile and delicately poised organ, and it has seemed to me probable that this response might have been due to movement communicated to sensitive portions of this organ.

My results are not in harmony with those obtained by Ivy and Johnson, and I am not able to explain the significance of the difference in the two sets of cases. Of course in any case of tumor it is possible that some nerve fibers are preserved, but this explanation would not apply to a case of nerve section subsequently verified by autopsy.

Postural Sense (Sense of Position and Passive Movement)

Is the afferent system concerned with the recognition of position and passive movement represented in the seventh nerve? This is a question which might receive an affirmative reply if the views of Head regarding the third or deep sensibility system are correct. Some consideration must be given to the position and relations of the seventh nerve musculature in beginning the discussion of this branch of our subject. The postural sense or sense of position and passive movement is both a muscular and arthroidal sense. Whether the latter can be separated from the former in its definition may be regarded as doubtful. The recognition of the position of a limb or a part of a limb is at least largely dependent upon impressions received from joints and the parts immediately controlling arthroidal movements. The face has but one joint under the control of the will, that which is concerned with the movement of the lower jaw, up and down as in opening and closing the mouth, and from side to side as in grinding movements. This movement is affected by muscles supplied by the motor fifth nerve, by the masseters, temporals and pterygoids. Other facial and ocular movements, while of much importance in expression, nasal respiration, articulation, and a few other well-known functions, are probably not to any large degree dependent for their proper performance upon the postural sense—at any rate not to the same extent as joint movements. The postural sense might therefore, it would seem, have a considerable representation in an afferent system passing with the motor fifth, but one much less significant as regards the seventh nerve. Nevertheless it is probable that there is some postural sense connected with facial movements, although this may not be of much importance.

Cushing, Krause and others have observed in cases of section of branches of the trigeminus or of its sensory root, or of extirpation of the gasserian ganglion, appearances and conditions in the face which seem to them to indicate that in some way the cutting of the fifth nerve root or its branches gives rise to some postural deformity. Even Sir Charles Bell as cited by Cushing observed some flattening and other changes in the contours of the face after section of peripheral branches of the fifth. Cushing has made similar and more extensive observations.

" In the period immediately following the neurectomy," says Cushing, " there may be seen, more marked in some cases than in others, a not inconsiderable hemilateral flaccidity of both upper and lower lips and possibly, as Krause has remarked, a slight flattening of the ala nasi and lessening in depth of the nasolabial fold, a condition somewhat emphasized by the temporary fullness of the tissues due to the vasomotor paralysis. At rest, with the lips closed, the lip margins on the operated side tend in some cases to remain slightly parted; during active movements the asymmetry between the two sides may become somewhat more pronounced, for example, in the effort to whistle when the lips may be satisfactorily puckered. In one of my cases for a few weeks the postural disturbance was so great as to simulate an actual hemifacial palsy, and only after electrical examination could it be proved otherwise. At a later period the flaccidity is frequently replaced by a corresponding degree of overaction in the expressional musculature, namely, by a slightly drawn position of the corner of the mouth, a deepening of the nasolabial fold, and in old people an accentuation of the wrinkles (crow's foot) radiating from the outer canthus."

Cushing speaks of the possibility of ascertaining the absence or presence of the postural sense in cases of trigeminal section by the use of the faradic current, stating that when the movements are strong enough to produce muscular contractions, these are not appreciated unless the twitchings are conveyed to an esthetic area. The patient also, he believes, does not appreciate passive movements produced in other ways, such as by hooking up the corner of the mouth or the ala of the nose.

In three of the cases of trigeminal section examined by me, I tested the patient in the manner here indicated, that is, both with the faradic current and also by using the finger or a bent aluminum probe as a hook with which to draw up the corner of the mouth or wing of the nose. When the areas investigated were totally anesthetic the patient did not appreciate the movement unless a very strong current was used. In one of the patients the current necessary to produce contraction of the levators of the angle of the mouth and nose and the dilator of the nostril was so strong as to be unendurable in any esthetic area of the face or limb. A minimal contraction was not appreciated, but when the contraction was so strong as to be easily visible to the observer, the patient spoke of feeling a drawing or pulling. I was not able to fully convince myself that this sense of contraction was not a result of extra-polar diffusion of the very strong current to neighboring areas which were esthetic or only partly anesthetic. The contraction or drawing was recognized, for instance, by two patients in whom the area of distribution of the second branch of the fifth was totally anesthetic, while the third area was only partially so. Such experiments as I have made are not sufficient to prove that the sense of passive movement was evoked through muscle sense nerves travelling with the motor facial. Cushing's view seems to be that the postural sense as recognized in the face is represented in the trigeminus.

I am inclined to believe with him that much of the postural asymmetry observed in the tongue, soft palate, and face is to be ascribed to section of the motor branch of the fifth, which usually accompanied operations on the sensory root, gasserian ganglion, or branches of the sensory fifth nerve.

Of course some of the postural asymmetry observed, both in peripheral neurectomies and in intracranial operations generally is due to the cutting of muscles and of branches of the facial or ocular nerves, or when these conditions are transient, to pressure on branches of the same nerves.

Little can be determined with regard to the existence in the distribution of the seventh nerve of afferent fibers concerned with the muscle sense by examination of cases of facial paralysis. The patients of course have a feeling of loss or impairment, but it is doubtful whether this is to be classed as a muscle sense impairment or sensation. Owing to the presence of sensation in the skin and mucous membrane a test with the electrical current and by placing the mouth or nostril in different positions can not be applied with any satisfactory results.

It has seemed possible to me that even if the afferent fibers concerned with the sense of pressure and of position and passive movement pass with the motor facial in the periphery, they may on entering the cranium diverge and join the sensory branches of the trigeminus before reaching the gasserian ganglion. It is well known that a considerable number of anastomoses take place between the facial and trigeminus.

Epicritic and Protopathic Sensibility and the Facial Musculature

The evidence of embryology, anatomy, surgery and medicine is conclusive that epicritic and protopathic sensibility (superficial touch, warmth and coolness, tactile discrimination, extremes of heat and cold, and pain) have no general representation in the facial nerve, and it would be scarcely worth while to consider this question were it not for the fact that occasional cases of facial paralysis are observed in which the patients are apparently slightly hypesthetic not only over the entire face, but sometimes in adjacent portions of the neck and head. In one typical case of facial paralysis studied by me, the patient as the result of numerous examinations uniformly declared that while he felt the lightest touch or prick on all parts of the auricle or behind it, he did not feel these impressions as well as on corresponding parts of the sound side. In two other equally typical cases, the patients in response to the examination said that they could feel better all over the face and on the tongue on the unparalyzed side. In all the other cases of facial paralysis, eight in number, the patients declared that touching, pricking with a needle or pin, pressure with the finger or other object, and various degrees of heat and cold were felt the same on both sides, with but two exceptions. In one of these exceptional cases the patient was slightly hypesthetic on the paralyzed side of the tongue, and in the other tactual stimuli were felt less in a very limited portion of the external auditory meatus. I was confident that in those cases in which there was a widely distributed light hypesthesia this was of a suggested or so-called hysterical character. One of the patients was a Polish Jew, another was a Russian Jewess, and a third was a colored woman, and all were distinctly of a well-marked hysterical or suggestible temperament. The patient who had the very slight and very limited hypesthesia of the external auditory canal was a woman and also, I believe, of this temperament. The man with the hypesthetic half of the tongue was an alcoholic but recently recovered from a prolonged debauch, during which this facial paralysis occurred.

SENSIBILITY OF THE PALATE

By a few observers it has been thought possible that sensibility was furnished to the soft palate or a portion of the soft palate from the facial by way of the geniculate ganglion and the great or small superficial petrosal nerve. Dixon, for instance, states that several observers have recorded cases in which sensibility was retained in the soft palate after section of the fifth nerve trunks. According to him the explanation for this may be found either in the sensory innervation from the seventh through the great superficial petrosal, or from the glossopharyngeal through the small superficial petrosal, more probably by the latter route. Dixon holds that the small superficial petrosal on embryological ground is certainly to be regarded as an afferent nerve, and no observations are forthcoming which show that the nerve contains taste fibers, and he therefore argues that it may contain fibers concerned with common sensibility. It is true, as he indicates, that in some cases of intracranial operation the soft palate loses its sensibility, but this he would explain not by the cutting of the fifth, but rather by the severance of the small superficial petrosal (a branch of the glossopharyngeal) which is in intimate relation with the dura in the middle cranial fossa.

My own observations appear to show that no part of the soft palate or nasopharynx is supplied with sensation by the seventh nerve. With Dixon I also believe that the great superficial petrosal is one of the primary branches of the geniculate ganglion, and is a nerve of taste, to the soft palate, not a nerve of common sensibility or a motor nerve. Later I shall speak further of the small superficial petrosal.

In the course of my investigations I examined or had examined for me by surgeons and laryngologists more skilful than myself, and usually in my presence, the nose, eustachian opening, soft palate, nasopharynx and fauces for sensibility both in the trigeminal and seventh nerve cases.

Sensibility was retained in all parts of the mouth and throat in all but one of the cases of facial paralysis. This case is deserving of especial detail. Evidently the loss of sensation was not due to any involvement of sensory fibers of the seventh nerve.

The patient was a young woman twenty-four years old, who had had a very severe attack of diphtheria one month before coming under observation. She had been out of the hospital about ten days, and four or five days before she was seen, she noticed stiffness in her face and neck and difficulty in swallowing. She had regurgitation of liquids through the nose when attempting to swallow. She had no pain of any character. She also had a feeling of numbness (rather indefinite and uncertain) in the right side of the face, and was unable to close the right eye or move this side of the face.

Examination showed a typical case of right-sided Bell's palsy, the upper and lower distributions of the nerve fully involved. Taste was lost in the chorda tympani area of the tongue. Faradocontractility was retained at the time of this first examination. All parts of the ear, face and tongue were tested for light touch, tactile discrimination, heat and cold, but with negative results—sensation was everywhere retained.

Examination by a member of the laryngological staff of the hospital of the University of Pennsylvania of the mouth and throat, including the nasopharynx, the opening of the eustachian tube, investigated by way of the nose, gave the following results: " Sensation was normal on the left or unaffected side; on the affected side sensation was much lessened around the eustachian tube opening. It was normal in the middle of the nasopharynx The faucial arch was relaxed on the right side and sensation was lessened. The right pillars showed reduced sensation; also the right pharyngeal wall. The muscles of adduction were weak in their action, and during quiet respiration the right side of the larynx was more relaxed than the left."

The impairment and loss of sensation in this case were probably to be attributed to glossopharyngeal and possibly also some vagal or fifth nerve implication.

In the examination of the buccal and pharyngeal cavities in the trigeminal neurectomy cases no special areas of anesthesia or hypesthesia referable to the seventh nerve were found. My own observation regarding the field of anesthesia in cases of sensory root section, gasserian extirpation or section of the second and third branches of the fifth, are practically in accord with those of Cushing as expressed in his admirable paper on the sensory distribution of the fifth cranial nerve. Dr. Cushing, who kindly placed at my disposal two of his cases, examined the tongue, mouth and throat of these patients in my presence with results which correspond to those which are given in his paper.

With regard to the eustachian opening and tube my own observations would seem to indicate that it has in minor part a glosso-pharyngeal sensory supply.

(To be continued)

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THE SEVENTH NERVE AND COMMON SENSIBILITY IN THE TONGUE

One region to which nerves concerned with some forms of common sensibility have been supposed to be distributed through the chorda tympani is the anterior portion of the tongue, but personal observations, especially some of those made in cases of peripheral facial paralysis, are mainly in opposition to this view.

"After a trigeminal neurectomy," says Cushing, "that portion of the tongue anterior to the circumvallate papillæ is an exception to the rule of total anesthesia to all forms of stimuli. Simple tactual impulses produced with a horse hair, as well as those of pain and temperature, are completely interrupted exactly to the mid line. Certain forms of common sensation are, however, preserved. If a cotton swab or a wisp of cotton be moved over the anesthetic field, its presence is recognized, localized more or less accurately, and its direction of movement appreciated. There are reasons for believing that certain fibers from this area of the tongue not only as the special sense fibers of taste but also of common sensation, pass to the brain by way of the chorda tympani and the nervus intermedius (Wrisberg). . . . In none of these cases was taste affected in the slightest by the operation; in all of them there was retained over the anesthetic portion of the tongue this peculiar form of sensitiveness to certain tactual stimuli. An examination, furthermore, of a number of cases of facial palsy, has shown that when the lesion is high enough to interrupt the transmission of gustatory impulses there is an accompanying slight sensory disturbance over the anterior portion of the tongue to these particular forms of stimuli, even though a normal sentiency to touch, pain and temperature seems to be present. Thirdly, the good fortune of being enabled to observe a patient in whom both the fifth and seventh nerves were totally paralyzed has shown that no sensation whatever over the anterior part of the tongue remained to touch, taste, pain, temperature or to the movement of the swab over the surface. These observations have been sufficient to establish from a clinical basis a personal conviction that there exist afferent fibers of two sorts, common and special sense fibers, which pass from the tongue via the chorda tympani and N. intermedius. It may be seen that morphological studies are in agreement with this."

I have already indicated when speaking of the pressure sense that the sensation apparently present in the tongue in Cushing's trigeminal cases might perhaps be explained by the movement communicated to this organ in drawing the swab or other object over it. Nevertheless in the light of his well-observed cases Cushing's view as to the anterior portion of the tongue being supplied in part with sensibility through the seventh must be given much consideration.

Three of the ten cases of peripheral facial paralysis studied by me showed some apparent change in sensibility. In two of these cases the hypesthesia was to be classed as due to suggestion, to belong in other words in the domain of hysterical hypesthesia. These were the same cases which showed a hypesthesia or apparent hypesthesia all over the face and the auricular region. This question of hypesthesia suggested by the presence of a motor paralysis must always be borne in mind in studying cases of facial paralysis for impairment of sensation.

In a third case, a man who was admitted to the hospital in a state of alcoholic stupor, the examination for sensation was made immediately after he had come out of this condition and when his mind was becoming alert. Testing the tongue with light touches to the right and left of the median line, in a ratio of four times out of five he said that he felt the touches more on the left than on the right or paralyzed side, while in testing for pain he responded that this was the same on both sides.

With regard to the condition of the tongue in the three trigeminal cases examined, the results were as follows: In one case in which an old complete section of the third branch of the fifth had been made, there was no sensation on the right (operated) side of the tongue to the hair test. The sensation to pain and temperature was reduced, not absent. In other cases where the third branch had been completely divided, sensation was entirely lost on the operated side.

The following note from Dr. George B. Wood, of Philadelphia, reporting the effect on the tongue of cocainization of the chorda tympani in the tympanic cavity, is of interest in connection with the discussion of this question.

"In an intelligent patient, a woman about twenty-five, a suppurative condition of the ear had destroyed the scutum so that one could see directly into the attic of the tympanum containing the bodies of the malleus and incus and other important structures. The drum itself was practically normal. On cocainizing the attic for the purpose of treatment, the patient complained of numbness on that side of the tongue, and on testing with a pin the sensation on the right anterior half of the tongue was apparently entirely lost, the remaining part of the tongue being normally sensitive. The explanation, of course, is that the chorda tympani nerve in its passage through the tympanic cavity became anesthetized from the cocain. Sensation returned to the tongue as soon as the anesthetic wore away."

THE VIEWS OF HUNT AS TO THE FUNCTIONS OF THE GENICULATE GANGLION SYSTEM

According to Hunt a strip of the external auditory canal and a portion of the concha, helix and antihelix have their sensory innervation (epicritic and protopathic) through the intermediary nerve of Wrisberg and geniculate ganglion. Three or four valuable papers by this author deal with facial (seventh nerve) paralysis associated with herpes of the cephalic extremity. A fourth paper is concerned with the subject of otalgia, particularly with reference to the part played by the geniculate ganglion and its branches in this affection.

Hunt's main contentions may be summarized as follows: (1) The geniculate ganglion and its proximal and distal branches are parts of an epicritic and protopathic sensory system, the geniculate ganglion being the homologue of the dorsal spinal ganglia and the area of distribution of sensation being as just stated, in the external auditory canal, concha, helix, etc. (2) The geniculate ganglion plays only a very minor rôle as the ganglionic center of an afferent gustatory system, Hunt's exact words being, " Save for an uncertain relation to the taste fibers of the chorda tympani the sensory mechanism of the facial has played no rôle in symptomatology, and has not had attached to it any definite sensory functions." (3) The geniculate ganglion is at times the seat of " herpetic " inflammation, with an eruption of herpes in an auricular cutaneous strip, the syndrome in some of these cases of geniculate posterior poliomyelitis being facial paralysis with herpes, in others facial paralysis with impairment of hearing or other acoustic phenomena and herpes. (4) Besides the herpetic eruption referable to lesion of the geniculate ganglion, herpes due to posterior poliomyelitis of other ganglia as of the cervical, the petrous, the jugular, and the gasserian, is sometimes associated with facial paralysis or with facial paralysis and auditory symptoms. (5) Otalgia, either idiopathic or reflex, is often to be referred to lesion or disturbance of the geniculate ganglion.

Hunt supports his position by reference to the embryological work of His and others, to some cases of neurectomy of both the fifth and the seventh nerves, and to a study of the various paralytic herpetic syndromes of the cephalic extremity.

The Geniculate System and the Sense of Taste—Embryological Teachings

In order that the views of Hunt receive acceptance one must be able to demonstrate in the first place, that the geniculate ganglion, as has been hitherto maintained by many competent observers, with its branches and central root, is not given over to the sense of taste.

One can admit with Hunt the homology between the dorsal spinal ganglia and the cranial ganglia—gasserian, petrous, jugular, and geniculate—without acknowledging that the functions of these ganglionic systems are the same as those of the spinal ganglia with their afferent nerves.

Let us now turn to this question of homologies as indicated by embryology. In the process of evolution the geniculate ganglion, and to a large extent in all probability the petrous ganglion, has become transferred in its functions to the sense of taste. Possibly some vestigial cutaneous afferent fibers may be present in the geniculate system, but this is all that with the evidence can be asserted.

Dixon, for instance, holds that in fishes and in lower air-breathing vertebrates the palatine and prespiracular nerves take origin together from the ganglion which is the homologue of the geniculate ganglion in man, while the post-spiracular on the other hand corresponds to the efferent motor facial. He shows that the great superficial petrosal nerve in man corresponds to the palatine, and the chorda tympani to the prespiracular, these nerves both in lower animals and in man going to regions which are especially concerned with the function of taste. The limits of this article will not permit a discussion of the details on which

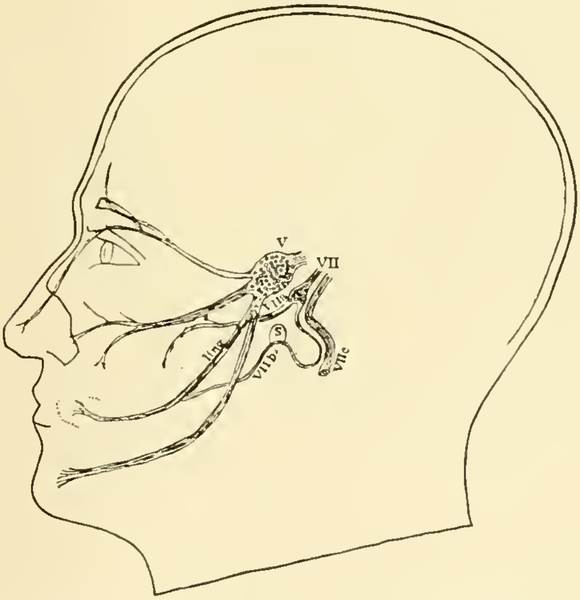

Fig. 1. Diagram to illustrate the sensory distribution of the facial nerve in man (Dixon). The sensory functions of the seventh nerve as here indicated are gustatory. The great superficial petrosal nerve (VIIa) is a nerve of taste from the soft palate to the geniculate ganglion. The chorda tympani nerve (VIIb) is the nerve of taste from the anterior two thirds of the tongue, except its tip, to the geniculate ganglion. The motor facial is indicated by VIIc. The gasserian ganglion and the three branches of the fifth are shown in the diagram.

these opinions are founded, but they certainly agree better with the idea of the gustatory functions of the chorda tympani and great superficial petrosal than with that which would make these nerve branches largely concerned with sensation in the skin and mucous membrane.

In most of the discussions of the question of the existence of areas in which common sensibility or the sense of taste has been referred to the seventh nerve, the great and small superficial petrosal nerves, especially the former, have played a considerable part. These nerves run between the geniculate ganglion and the sphenopalatine and the otic ganglion which are generally regarded as belonging to the fifth nerve.

It will not be necessary to recall here the well-known description of the course of the great superficial petrosal after it leaves the geniculate ganglion. It is sufficient to say that in a certain part of its course it joins with the great deep petrosal nerve to form the vidian, through which its course is to Meckel's ganglion. One of the descending branches of Meckel's ganglion is the posterior or small palatine nerve which is distributed to the soft palate.

According to some physiologists the great superficial petrosal is an afferent nerve with sensory functions; according to others it is motor; while others regard its trunk as conveying both afferent and efferent fibers. By some its afferent functions are held to be gustatory; by others sensory in the ordinarily accepted cutaneous meaning of the word. Even those who have regarded the great superficial petrosal as a part of the gustatory pathway have differed with respect to the direction in which the impulses concerned with taste travel in the nerve.

Presumably if the great superficial petrosal is an afferent nerve, the anatomico-physiological pathway would be from one of the branches of Meckel's ganglion, the posterior palatine for instance, to this ganglion and thence by way of the vidian to the great superficial petrosal, the geniculate ganglion, and the pars intermedia to the nuclear terminus of this nerve. If this view be accepted the great superficial petrosal nerve can take no part in auricular cutaneous sensation or otitic herpes.

Returning now to the question of homologies, arguments based on these, especially when such reasoning derives its chief strength from embryological and histological studies, are of great value, if the facts are properly interpreted. Dixon on his part and Hunt on his, both use such arguments, and yet arrive at quite different conclusions with regard to the geniculate ganglion. In the case of the jugular and petrous ganglia it is probable that some of the afferent fibers and roots springing from these bodies are concerned with the common sensibility (cutaneous sensibility, epicritic and protopathic). In the case of the petrous ganglion a large portion of the cells of this ganglion are regarded with little opposition as having gustatory functions, the root fibers going to the glossopharyngeal nucleus or a part of it, and the peripheral fibers to the back part of the tongue and other buccal surfaces. In the case of the ganglion of Scarpa on the vestibular nerve, and the spiral ganglion of the cochlear nerve, the afferent systems of fibers are concerned with equilibration and orientation and with hearing, not with cutaneous or membranous sensibility. The geniculate ganglion has for the termination of its root, the pars intermedia, the upper part of the nucleus of the glossopharyngeal, and for its peripheral portions the chorda tympani and the great superficial petrosal nerves, and the weight of the evidence is entirely in favor of this afferent system being concerned with the function of taste.

It must be remembered here that only those fibers, probably vestigial if they exist at all, which are concerned with the innervation of cutaneous surfaces, would be likely to be involved in a disease with cutaneous manifestations.

Similarly the ganglion of Scarpa and the spiral ganglion of the cochlear nerve, while they may be homologous with the spinal ganglia, are concerned with non-cutaneous functions, and can take no part in a cutaneous disease. Hunt is right when he says that these ganglia may all take part in an infectious inflammatory disease which in the case of the spinal, gasserian and other ganglia gives rise to herpes. The results of such disease would not, however, be an herpetic eruption, but in the case of the geniculate ganglion would rather be a syndrome mainly gustatory, in that of the ganglion of Scarpa symptoms mainly of equilibration and orientation, and in that of the ganglion of Corti acoustic phenomena.

Surgical Observations Regarding the Course of the Nerves of Taste

In the cases of section of the fifth nerve studied by me with regard to the loss or preservation of taste, the evidence is opposed to the views of Gowers and others who believe that the taste fibers of the chorda tympani distribution run to the gasserian ganglion and pons by way of the fifth nerve, my observations being entirely in accord with those of Cushing respecting this much disputed matter. It is true that in some instances in my cases taste was lost after trigeminal section in an early period, one usually of only a few days and always less than two weeks, but was gradually or somewhat rapidly restored. In some of these cases the gustatory response when present was more delayed upon the paralyzed than upon the unaffected side. Doubtless the explanations offered by Cushing for this partial and early loss of taste are the best, namely that interference with the vascular supply and the nutrition of the tongue which results from the fifth nerve section has a considerable effect for a short time upon the taste buds, although the exact mechanism of this interference is not easy of explanation. This abeyance of the sense of taste may explain the cases which have been reported by such highly competent observers as Gowers and Horsley.

Loss of Taste in Peripheral Facial Paralysis

The cases of peripheral facial paralysis now reaching into scores or even hundreds studied by me during a long neurological practice, when severe if carefully studied have almost invariably shown impairment or loss of the sense of taste in the well-known chorda tympani distribution on the anterior portion of the tongue. My observations as to the preservation or impairment of taste in the palatal region are much less numerous, but are sufficient to indicate that loss of taste does not commonly occur.in Bell's palsy. That taste buds are present in the soft palate need not be demonstrated. The evidence is abundant with regard to this matter. In the two cases of Hunt's first paper on "Herpetic Inflammation of the Geniculate Ganglion, a New Syndrome and Its Complications," he says that taste was not lost. Two explanations may be offered of this finding. The first case was one of very mild type, recovering in three or four weeks, and in such cases occasionally the impairment of taste is slight, transient, or not present. The second case, in which he obtained a necropsy, was of serious type, and the only explanation which I can offer is that it is possible the gustatory response which he obtained was from the extreme anterior part or tip of the tongue on the affected side. This small area, as I have shown elsewhere, is supplied not by the chorda tympani but by a branch of the glossopharyngeal. In all the cases of facial paralysis, especially studied in the preparation of this paper, taste was lost in the chorda tympani area, in one case on both sides of the tongue, a matter difficult to explain unless it was dependent on some local condition.

Vasodilator and Secretory Fibers Which Pass by Way of the Geniculate Ganglion

Brief reference should be made in passing to the fact that experiments which are regarded as almost classical in physiology have demonstrated that through the chorda tympani pass vasodilator and secretory nerves, the former to the tongue and probably to the sublingual and submaxillary glands, and the latter to these glands. These vasodilator fibers, however, while passing by way of the geniculate ganglion, have no direct connection with it, and the secretory fibers for the sublingual and submaxillary glands have ganglia of their own in or near these glands.

Supposed Pathway of Sensory Impressions from the External Ear to the Geniculate Ganglion

Leaving now the questions of the gustatory, vasodilator and secretory functions of the nerves of the geniculate system, let us next turn to the cutaneous sensory supply of the external ear. Taking up the question first, of the route by which the auricular sensory area is supposed to be connected with the geniculate ganglion one pathway that has been suggested has been through the auriculotemporal distribution of the fifth to the otic ganglion, and thence by the small superficial petrosal to the geniculate ganglion. Of the small superficial petrosal nerve it may be said that both its anatomical and its physiological relations are not as yet clearly defined. Following the usual anatomical descriptions, this nerve originating in the geniculate ganglion soon leaves the fallopian aqueduct through a canal near the hiatus fallopii, and goes forward lying in an osseous groove under the dura, usually escaping from the skull by a small foramen to entér the otic ganglion. This ganglion has traversing it branches from the fifth, probably motor, to the tensor palati and tensor tympanum, and also according to received anatomy, it gives branches to the auriculotemporal nerve and to the chorda tympani.

If the geniculate ganglion is supposed to be the origin of the cutaneous and membranous supply of the tympanic membrane and the skin of the external canal and auricle through the small superficial petrosal and otic ganglion, it could only be in this roundabout way.

Another course, however, may be taken by the small superficial petrosal nerve after it leaves the geniculate ganglion. It is usually said to give off a branch to the tympanic nerve, but some regard this as the main division of the nerve; still others regard this as a branch of the tympanic nerve of the glossopharyngeal to the lesser superficial petrosal nerve. If sensory impressions are conveyed from the membrane of the tympanum, external auditory canal, and auricle by way of the small superficial petrosal nerve, it would seem that it must be through this communicating branch between the tympanic branch of the glossopharyngeal, or by way of the communicating branches of the otic ganglion to the auriculo-temporal or the chorda tympani. Such sensory impressions may of course take their way as thus indicated, but this is a mere matter of conjecture, and has not been proved by anything that has been reported.

The Fifth Nerve Supply to the Ear as Determined by Sections of the Trigeminus

In those cases of fifth nerve section or gasserian extirpation studied by me in which either the third branch of the fifth or both the second and the third branches of the fifth were directly or indirectly severed, the fifth nerve supply indicated was to the tragus and anterior part of the canal, much the same as that determined by Cushing. After trigeminal section this region of the ear is not insensitive to all forms of sensation. It may respond to painful stimuli, but not to examination with the hair esthesiometer or with a small camel's hair brush. These findings are in accord with those of Cushing who found a narrow strip of lost tactual sensibility bordering his large general area of complete anesthesia after trigeminal section.

It is probable that in the compacted space which forms the external auditory meatus the overlap of the different neural supplies to the ear—the trigeminal, great auricular (cervical), vagal and possibly the glossopharyngeal—is such that section of any one of the nerves going to the auricle will not cause complete anesthesia. As determined by Head in his investigations on the median and other nerves if one nerve is cut, as the median, the area of complete protopathic loss is smaller than that for the other forms of sensibility, protopathic sensibility being preserved in the area of section through overlap of the adjoining nerve supply, in the case of the median for instance, by ulnar overlap.

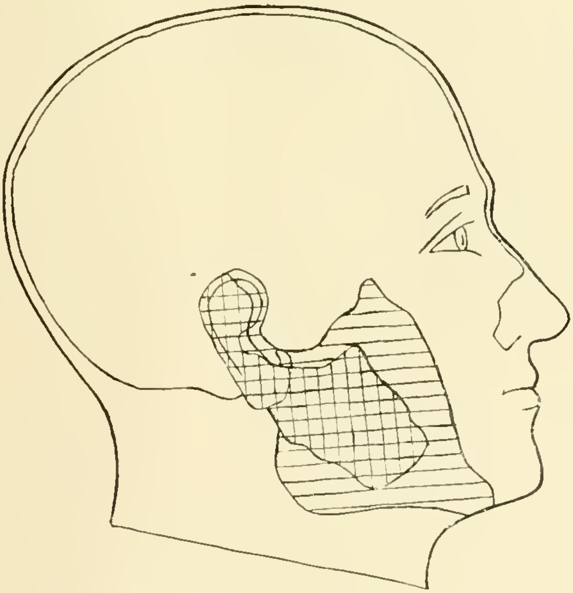

FIG. 2. Tactile changes following section of the great auricular nerve. Examination twenty-four hours after section. Vertical shading denotes anesthesia to camel's hair brush; horizontal shading denotes area abnormal to stroking touch. (Trotter and Davies.)

Great care should be taken in drawing conclusions in cases of supposed gasserian extirpation or of section of the sensory root of the trigeminus. Not infrequently this extirpation is incomplete and it sometimes happens that the sensory root is not cut or only partially sectioned when it is believed that a complete section has been made.

Distribution of the Great Auricular Nerve to the External Ear as Determined by Section of this Nerve

The distribution of the great auricular nerve to the ear and adjoining parts as demonstrated by section of this nerve in man, is indicated in a diagram in one of the contributions of Trotter and Davies. These investigators found that twenty-four hours after section of this nerve, which was performed on one of them for the purpose of studying the conditions of sensation after section and during the repair of nerves, impairment of tactual sensation was present in the areas indicated in the diagram. The more marked anesthesia was in the more central portion of the area, including the ear, and was determined by the camel's hair brush. This is indicated by the vertical shading. A wider limit of slighter hypesthesia was determined by the finger stroking method and is indicated by the horizontol shading.[2]

Distribution of the Vagus to the External Ear

The auricular branch of the vagus as described in Morris' anatomy by Dr. H. St. John Brooks, has the following course and distribution: "It arises from the jugular fossa, and traverses the bone, passing to the inner side of the aqueduct of Fallopius, and merges behind the pinna, where it divides into two branches, one of which joins the posterior auricular branch of the facial, while the other supplies the posterior and inferior part of the external auditory meatus and the back of the pinna."

This auricular branch of the vagus is to be regarded as afferent (cutaneous, sensory), and as having its nuclei of origin in the jugular ganglion. It is very rare to get a case of any form of disease which will throw light upon the distibution and the function of this branch of the vagus.

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[2] In the finger stroking method, which was first suggested by Trotter and Davies, the patient examines himself for impaired sensibility by gently moving the finger tip or tips from the regions of retained sensibility toward that in which it is presumably impaired. As soon as this limit is reached he will usually at once indicate the fact. The method has proved in my hand a very useful one, the patient frequently marking out with great exactness a region of very slightly diminished sensibility.

Some well-known facts with regard to the reflexes from the external auditory canal are not without value. In many persons a cough is at once produced by slight irritation of the external auditory canal. This often occurs during an examination of the ear. I have made some personal observations with regard to this reflex which are in accord with those which have been reported to me by some of my friends among the otologists and laryngologists. Dr. George C. Stout and Dr. B. A. Randall, of Philadelphia, for instance, are both inclined to place the seat of the most ready production of this aurolaryngeal reflex in the posterior inferior portion of the auditory canal. In some, slight irritation of this region by a probe or light object of any sort may cause a sharp cough difficult to control. This region of easy stimulation is probably within the area of the vagus.

Besides the supply to the posterior wall of the canal the auricular branch of the vagus probably also is distributed to a portion of the concha, and to the postero-mesial surface of the auricle; possibly also to the posterior segment of the tympanic membrane, although this is supposed by some to be in the distribution of the glossopharyngeal.

Hunt in his extensive research of the literature of herpetic inflammation of the cephalic extremity was able to find only one case, that of Buys, which he supposed might be referable to involvement of the ganglion of the vagus. "A girl aged seventeen years. Onset with headache and vomiting. Severe headaches continued with stiffness of the neck and frequent vomiting. Photophobia, no fever and no delirium. The pulse was slow and irregular. On the fourth day the acuteness of the pain subsided and settled in the mastoid region of the right side. The mastoid was tender, as was the canal on the introduction of the otoscope. Hearing was diminished. On the fifth day herpetic vesicles made their appearance on the antitragus and lobule of the ear. The headache and neck pains were less severe. On the sixth day a fresh crop of vesicles made their appearance on the mesial surface of the pinna and the lobule. All pain disappeared in the course of a few days, and the hearing was restored. In fifteen days the eruption had vanished." Some of the symptoms in the case were distinctly vagal.

According to the description of the auricular branch of the vagus as generally accepted, the herpes of the membrane described by Orbison might be referred to this nerve as readily as to the glossopharyngeal. Indeed this area may be in whole or in part supplied either by the vagus or the glossopharyngeal, or by both, these two nerves arising in adjoining ganglia.

The Teaching of Cases of Auricular Herpes

Such cases of herpes of the cephalic extremity as I have seen, as well as the literature of the subject, point to the herpetic inflammation having occurred either in the gasserian ganglion, the ganglia of the second and third cervical nerves, and possibly the petrous or the jugular ganglia. In the vast majority of cases the evidence is in favor of the cervical and the gasserian ganglia. An otitic herpes of geniculate origin seems to be largely presumptive. The herpetic ulcers in all the cases described have been either on the auricle itself or in the external auditory canal—in a very few instances like that reported by Orbison, on the membrane of the drum.

The following case of herpes is of interest as bearing upon the question under consideration, that of the nerve supply to the external ear.

A man about seventy-three years old was seen in consultation with Dr. J. Gurney Taylor. The patient was in good general health, active for his age, but for several years at times had suffered with attacks of pain with some swelling about the right ear, seemingly just outside and at or near the entrance to the external auditory canal. Shortly before he was seen by me he had a sudden and very severe attack of herpes. His face along the lines of eruption was extremely painful and much swollen. After the violence of the first attack, in a day or two, the generalized inflammation began to subside. It was then found that he had a widely distributed region of herpetic ulcers.

At the time of my examination these ulcerations and the inflammation attending them had largely subsided. The herpes covered a large area of the posterior part of the head, neck, the lower face, and the ear and region in front of it. The eruption was especially marked and widely distributed on the ear. One ulcer was present at the entrance of the external auditory canal at its posterior inferior part. The patient complained of a feeling of impaired sensation, and examining him with a finger and with a hair over the head, ear, and face, he did not feel the touches and manipulations quite as well on the eruptive as on the other side. He was nowhere totally anesthetic, and in various places he still had pain.

It was evident that the nerve distributions indicated by this eruption were confined to the upper cervical nerves, particularly the second and third. The trigeminus and vagus were not implicated. It was not necessary to evoke either the petrous or the geniculate ganglion to explain the eruption.

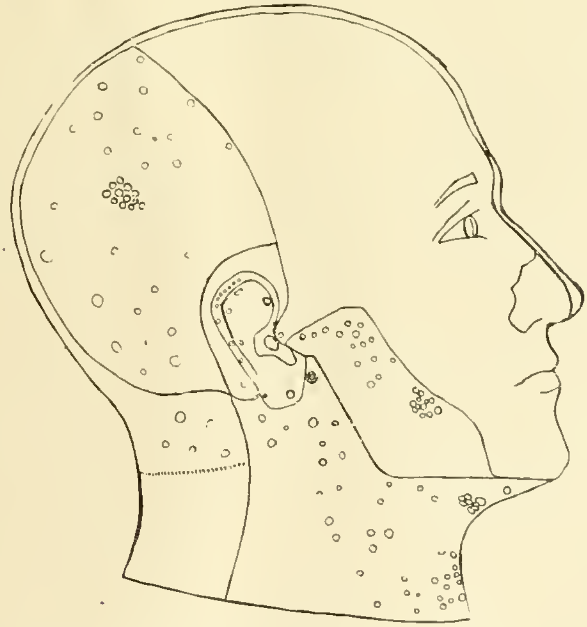

Fig. 3. A case of herpes occipito-collaris indicating by the eruption the superficial sensory distribution of the upper three or four cervical nerves. The areas included are those of the great and the small occipital, the great auricular and the superficial cervical nerves. The trigeminal sensory distribution is outlined exactly by its exclusion from the eruption.

Distribution of the Glossopharyngeal Nerve to the Tympanic Cavity, Membrane and External Auditory Canal

The branches of the glossopharyngeal which are sometimes given as going to the membrane of the tympanum and external auditory canal, as well as to the palatine and alveolar regions, arise in the petrous ganglion (ganglion of Andersch). The branch which passes from this ganglion to the tympanic cavity is known as the tympanic nerve or the nerve of Jacobson. It has a half dozen branches, which with their communications form the tympanic plexus. A portion of the tympanic membrane, probably its posterior segment, may be supplied by one of these branches of Jacobson's nerve. Other branches, as has already been shown, communicate with the great and the lesser superficial petrosal nerves, and in the case of the small superficial petrosal, go to the otic ganglion, and by way of the great superficial petrosal and vidian branches may proceed to Meckel's ganglion and thence to the palatal region. It is known that the branches of the tympanic plexus supply the mucous membrane, or a part of it, lining the membrane of the tympanum, and also of the eustachian tube.

Orbison has reported a case which is of value and interest in connection with the part of our problem under discussion. The case was well observed, the herpetic bleb on the membrane of the tympanum not only having been studied by Orbison, but by a laryngological colleague, Dr. Roberts. Briefly summarized, this patient, who had a gouty and rheumatic history, and what might be termed both a personal and a family history of herpes, as one of her brothers had had this disease, and the patient herself had suffered from it on several occasions in various parts of the body, was attacked with a painful throat and naso-pharyngeal affection, probably rheumatic or gouty in origin. This was accompanied by constitutional symptoms and infection. On the third day she had earache and fulness in the left ear. Deafness soon followed. Aural examination showed a bleb on the left membrane of the tympanum, beginning in its posterior part and soon covering the whole membrane, and looking like a crystal. It is interesting to note that the use of the speculum caused but little pain, showing probably some anesthesia. In three days the bleb began to disappear and with it the deafness. Later eruptions were present in the mouth, and on the palatine and alveolar regions, and the patient had a polyneuralgia during her entire illness.

It might of course be claimed that the evident herpetic inflammation of the membrane in this case was due to the involvement of some other zoster zone than that of the petrosal ganglion. With regard to the geniculate ganglion however, it must be remembered that symptoms usually accompanying affections in this ganglion were not present unless we regard the tinnitus and deafness as such, but these are much more readily explained by the implication of the tympanic cavity and membrane. Facial paresis or paralysis was not present. It might be said in this connection that the symptom complex of herpes with acoustic phenomena might in most cases be quite as readily explained without as with involvement of the geniculate ganglion, and the adjoining auditory nerve. Deafness, tinnitus, pain in the ear, hyper-acousia, or hypo-acousia, one or all, may be produced by affections of the middle or even of the external ear. Is it reasonable, therefore, to tabulate such cases as if they were in all or a large percentage of cases due to herpetic inflammation of the geniculate ganglion? Often the symptom complex includes facial paralysis and auditory phenomena of various description and may of course be due to nerve affections at points removed from the geniculate ganglion. Herpes is just as likely to be the result of petrous, jugular or cervical ganglionic disease, as of disease of the geniculate ganglion.

One of two cases reported by Hunt, a case in which a necropsy was obtained, was a man forty-eight years old, who when admitted to the hospital was suffering from facial paralysis and herpes occipito-cervicalis (occipito-colaris). The symptoms were those of a case of typical peripheral facial paralysis with herpes in the distribution of the second and third cervical nerves. On necropsy the facial nerve, the intermediary nerve of Wrisberg, brain, spinal cord, etc., were removed. The geniculate ganglion was lost in the necropsy. The microscopical examination showed degeneration in the peripheral portion of the seventh nerve, in the intermediary nerve of Wrisberg, and some change in the nuclei of the facial. Similar degeneration was found in the cervical nerves, and certain changes in the cord. The chorda tympani was apparently not examined. If the geniculate ganglion was involved, the result was simply to give a syndrome such, with the exception of the loss of taste, as is usually obtained in any case of seventh nerve paralysis, the herpes being referable to the inflammation of the cervical ganglia.

THE QUESTION OF THE IMPAIRMENT OF SENSATION IN PERIPHERAL FACIAL PARALYSIS

The entire number of cases of facial paralysis included in this study were examined for light touch, and for pain in all parts of the external ear including the canal. In three instances, as already described, the patient was hypesthetic on the ear of the paralysed side, but this hypesthesia covered distinctly the areas of the cervical, fifth and vagus nerves. Indeed it included all parts of the ear and its posterior and anterior borders. In two of the cases the patients were hypesthetic over the entire side of the face and head and also on the paralyzed side of the tongue. Unquestionably these cases were instances of so-called hysterical or suggested hypesthesia. In only one case was there an extremely slight hypesthesia in a narrow strip at the entrance of the ear.

In the light of these cases I do not see how it is possible to conclude that there is a cutaneous auricular area which is supplied by the sensory branch of the seventh nerve.

THE GENICULATE GANGLION AND PAIN IN THE EAR

In the paper on otalgia considered as an affection of the sensory system of the seventh cranial nerve, Hunt collects an important array of well-known facts relating to otalgia, in support of his geniculate facial sensory theory. Accepting his view of the sensory functions of the geniculate, pain circumscribed to the ear is regarded as in the sensory division of the seventh. His claim is that in certain sharply circumscribed otalgias, the pain under the theory adopted can only be explained by the direct or indirect implication of the geniculate ganglion or its central or peripheral processes.

Little need be said about the hypothesis of the geniculate origin of primary otalgia. It amounts to little more than an assertion that this is due to geniculate disease.

The forms of otalgia of most practical interest because of their frequent occurrence and the numerous observations which have been made of them, are those which are classed under the head of referred and reflex otalgias. Here the otologist, laryngologist, dental surgeon, neurologist and internist all might have something to say from their experience. Briefly stated, Hunt's argument is that otalgia is a somewhat frequent referred symptom, and this no one will deny, occurring especially as the result of various diseases of the mouth, throat, teeth and jaws. In all such affections however the pain is referred from a region which is universally regarded as supplied by branches of the fifth, or in a few instances perhaps by the ninth or the tenth. According to this view when one has a pain in the ear from an abscess at the root of a tooth, from a sinus involving the periosteum and bone, from a catarrhal inflammation of the nasopharynx, in the disease focus originate impulses which are conveyed through branches of the fifth or other nerves by well-known routes chiefly petrosal, to the geniculate ganglion, and thus pain is produced in the auricle and external auditory canal, including the membrane of the tympanum.

This argument is at least not convincing. As the trigeminus, glossopharyngeal, and vagus all have a sensory supply to the ear, why is it necessary to evoke the geniculate ganglion, and the seventh nerve, in order to explain the pain? Is it not more reasonable to suppose that the pain impulses travel by routes forming a part of the neural distribution of the nerve in which they originate?

One case of operation for what was supposed to be idiopathic otalgia has been recorded by Clark and Taylor, of New York. In this case section of the posterior root of the geniculate ganglion (intermediary nerve of Wrisberg) was performed for the relief of pain. The case was reported very early after operation. It is held by the recorders that the relief of the pain in this case was due to division of the seventh-nerve's sensory root. The effects of decompression in such cases should not be overlooked.

Several years since Dr. Chas. H. Frazier, in one of my cases, after making the subtentorial intracranial opening, severed both the seventh and the eighth nerves as indicated by the complete facial paralysis and deafness. This patient had suffered intensely with pain in the head, not especially confined to the ear, although more marked on the side of operation. During the few days preceding the surgical procedure the pain was so constant and so intense as to call for the administration every two or three hours of narcotics and sedatives. The pain disappeared after the operation and has never returned, although the patient still has some symptoms of intracranial and cranio-neural disease. I was inclined in this case to attribute the result more to the effects of decompression than to section of the seventh and eighth nerve, and refer to the case as one of interest in connection with the general discussion of the sensory function of the accessory seventh.

A CASE OF PERIPHERAL FACIAL PARALYSIS WITH NECROPSY AND MICROSCOPICAL EXAMINATION

In one case of seventh nerve paralysis studied at the Philadelphia General Hospital, I had the opportunity of obtaining what is in my own experience extremely rare, a necropsy and microscopical examination. These were both conducted by Dr. Edward Mercur Williams, clinical and pathological assistant in the neurological department of the University of Pennsylvania. Dr. Williams also had the opportunity of studying this case with me.

This patient was a man eighty years old, who was admitted to the Philadelphia General Hospital, November 5, 1909, suffering from general debility, with advanced arteriosclerosis. Necropsy showed advanced disease of various organs, including cardiac degeneration, fibroid tuberculosis of the left lung, hemorrhagic cystitis, acute fibrinous peritonitis and carcinoma of the stomach. This man had a right-sided facial paralysis which was ten days old at the time of his death.

Examination immediately after his admission showed a complete seventh nerve paralysis. The patient was unable to wrinkle his forehead on this side, to close the eyelid, draw up the corner of his mouth, and his speech was difficult to understand, apparently because of the interference with articulation as a result of the palsy. He stated that three days previously, on awaking in the morning, he had for the first time noticed this paralysis.

Owing to the man's low intelligence or mental state it was difficult to obtain facts from him. No sensory changes were found on examination of the external ear and canal, and no area of hypesthesia on the anterior part of the right side of the tongue, although the mental and physical condition of the patient made it difficult to determine any facts regarding sensation and impossible as regards taste.

At the autopsy the facial nerve external to the stylo-mastoid foramen, with its several branches, to a distance of about an inch, was carefully dissected out. The skull was then chiseled and broken in such a way as to remove largely the petrous portion of the temporal bone with the internal and external auditory canals and the stylo-mastoid foramen, the peripheral portions of the nerve being kept intact. Because of the manner in which the bone was cut through, the tympanic cavity was exposed and a portion of the chorda tympani was removed for examination. By carefully nipping the bone from the internal auditory meatus, the course of the facial nerve was followed, and a portion of this nerve obtained, also the geniculate ganglion, a small part of the great superficial petrosal and of the proximal portion of the facial with the pars intermedia, although it was impossible to separate the facial from the intermediary nerve.

The geniculate ganglion stained by methods of Marchi and hemalum-acid fuchin, showed the cells to be, with few exceptions, normal in size and contour, having nuclei distinct, well stained and centrally placed. The layers of cells forming the capsules of the ganglion cells were also normal, showing neither faulty staining, disintegration nor proliferation. The amount of pigment in the cells was rather large, but not excessive and could not be considered abnormal in a patient of such advanced age.

A few of the cells, as mentioned above, showed a shrinking, with vacuoles around the periphery. These however were only postmortem changes, and the staining properties of the cells were otherwise normal, and the cell capsules were nowhere broken down. No cellular infiltration nor any other change indicated any acute or chronic process as occurring in the ganglion.

The facial nerve showed degeneration throughout its course from the part external to the stylo-mastoid foramen to and including the part entering the internal auditory meatus. This degeneration was, however, more marked and more extensive in the peripheral part. It was also very distinct in the part close to and at the level of the geniculate ganglion.

The part just at the entrance of the internal auditory meatus was degenerated, but not to the extent of that peripheral to this ganglion, that is, not as many fibers comparatively speaking, were degenerated as in the peripheral trunk.

The chorda tympani and the great superficial petrosal nerves were both normal; the latter were sectioned at its entrance to the geniculate ganglion, but showed no degeneration nor change of any character.

The intermediary nerve of Wrisberg could not be separated from the facial sufficiently accurately for study.

As bearing upon the main subject of this paper, the sensory functions attributed to the seventh nerve, this necropsy, although interesting, does not perhaps shed much light. It has, however, two or three points of value, even in this discussion. In the first place it showed probably the usual method in which the seventh nerve is attacked, and in which it degenerates in the average case of Bell's palsy. What probably takes place is that the nerve exterior to the foramen is attacked by the inflammatory process, this extending through the foramen into the fallopian canal. The geniculate ganglion is in all probability very rarely primarily attacked in the ordinary case of seventh nerve paralysis. It may, however, in particular instances be the seat of a posterior poliomyelitis as believed by Hunt. In such cases it is altogether probable that other ganglia concerned with cutaneous nerve functions would be the seat of similar inflammation and would be the pathological cause of any herpes that might be present. The chorda tympani in the case studied by Williams seems to have been not degenerated. This may be the case even in some of the instances of severe facial paralysis in which impairment or loss of taste is present on the anterior two thirds of the tongue. The chorda tympani in these cases becomes involved through pressure from the inflamed facial or perhaps by direct extension of inflammation to it. In some cases, however, the chorda tympani may largely escape serious interference with its functions, thus accounting for those cases in which taste is not at all or but little impaired.

Conclusion

1. If by the seventh nerve is meant that nerve trunk which arises in the pons from the lower and upper facial nuclei, this nerve is purely motor.

2. It is doubtful whether nerves whose functions are concerned with the pressure and postural senses are conveyed in the branches and trunk of the motor facial.

3. The afferent systems for epicritic and protopathic sensibility are not represented in the seventh nerve.

4. The geniculate ganglion is the homologue of the dorsal spinal ganglia.

5. If the geniculate ganglion, the intermediary nerve of Wrisberg, and the chorda tympani are considered part of the facial or seventh nerve, then this nerve may be regarded as partly sensory, having functions chiefly gustatory.

6. Nerve fibers concerned with the transmission of gustatory impulses proceed from their origin in the geniculate ganglion to their distribution by way of the great superficial petrosal nerve and the chorda tympani. The destination of the former nerve is the soft palate, where its fibers terminate in taste buds, and of the latter nerve the taste buds in the anterior two thirds of the tongue with the exception of its tip.

7. Evidence is lacking that in the facial trunk proper, in the intermediary nerve of Wrisberg, the great superficial petrosal, the small superficial petrosal, or the chorda tympani, are nerves of common sensibility, although some observations would seem to indicate that the anterior part of the tongue and possibly a very small strip of the auricle have a vestigial supply of this sort.

8. Herpes does not originate from inflammation of nerves which are not concerned with cutaneous or membranous sensibility.

9. Limited inflammation of the geniculate ganglion may in rare cases occur, producing a syndrome whose factors are loss or perversion of taste and vasodilator and secretory phenomena.

10. If the geniculate ganglion is the seat of inflammation, adjoining parts such as the facial nerve proper, and the eighth nerve may be involved by pressure or extension, thus giving rise to peripheral facial paralysis and to acoustic phenomena and phenomena of equilibration and of orientation, vasodilator and secretory symptoms or to some of these symptoms.

11. The cases of herpetic inflammation—cervico-occipital, auricular and facial—which have been observed in connection with facial paralysis, or facial paralysis and acoustic symptoms, are best explained on the supposition of an involvement of ganglia other than the geniculate.

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TUMOR OF THE FRONTAL SUBCORTEX AND CALLOSUM, WITH FLACCID PARALYSIS OF THE MUSCLES WHICH SUPPORT THE HEAD, APHONIA, MENTAL CHANGE, AND OTHER SYMPTOMS, ILLUSTRATED BY A CASE WITH NECROPSY[1]

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The following case is presented as a contribution to our knowledge of frontal, subcortical and callosal tumors:

Well marked general symptoms of brain tumor—inability to hold the head erect—aphonia—paraparesis, unskilful and uncertain movements and late right hemiplegia—exaggerated reflexes on the right side—involuntary evacuations—mental change—hypesthesia to pain and temperature—tumor involving the subcortex and frontal region of the left hemisphere and the callosum.

The patient, J. B., fifty-two years old, a manufacturer, was in good health with the exception of headaches until about two years before coming under observation. He began to suffer with these headaches at irregular intervals, and later had attacks of dizziness, and much more rarely of sickness of the stomach. He kept a diary, entries in which made during the period between January 9, 1905, and April 26, 1906, showed that he suffered not only from headache and dizziness, but also from rarer spells of nausea, and at times he had sensations of numbness or tingling or some form of paresthesia on his right side, especially in his right upper extremity.

On July 6, 1906, twenty-nine days before the operation, he had a severe spell in which he complained of his head and of great dizziness and plunged or fell towards the right side, going down upon the floor, but not becoming absolutely unconscious. After this he became much worse as regards his headache and general feelings of discomfort. Four days later he was taken to the Long Branch Hospital.

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[1] Read before a meeting of the Philadelphia Neurological Society, held November 27, 1908.

After his admission to the hospital he grew steadily worse although he had at times periods of moderate improvement with recrudescence of his old symptoms with additional severity. It was noted at the hospital that he tended at times to give way on the right or to fall in this direction, and that he stood and walked unsteadily as if both legs were somewhat weak.

When first examined by me at the hospital he was in bed, where he had been most of the time for some days, although he was taken out of bed daily and put on a chair. His evacuations were usually involuntary. His appearance of face was that of distress and confusion. He understood what was said to him when the matter presented was not too extended or complicated. He answered correctly various questions which were put to him, giving the names in succession of two sons when asked who they were, and also responding in other ways. His responses were always by " yes " or " no " or by one or two words only. He did not talk spontaneously and he did not appear to be able to form sentences of any length. The interne stated that a few days previously to the examination by me he had answered questions, using some short sentences correctly. His speech was not paretic or ataxic, but the little he said was spoken in a very low voice, or a whisper.

Examination of the eyes showed no paralysis of single or associated ocular movements, no ptosis, no difference in the pupils, which were of equal size and responded to light and to converging and diverging movements.

Examination for movements of the jaws and face showed no loss of power in the distribution of the fifth nerve, but drooping of the face on the right and slight impairment of volitional movements on this side. Both eyes could, however, be opened and shut. The patient could not be made to put out his tongue, although he moved it around inside of his mouth.

The right arm was paretic, the impairment of power seemingly being equally distributed throughout the extremity. The patient could, however, at this time use the hand and arm to some extent, as when stimulated to return a grip he grasped the hand of the examiner feebly. The left upper extremity was used with comparative freedom and force, both in response to grasp and other methods of examination, and also spontaneously. The left hand was frequently used to brush away flies, or the hand and arm were moved about in the restlessness and suffering of the patient.

The right lower extremity was distinctly paretic, the impairment of power here being also general, and was, on the whole, about as marked as in the upper extremity. He used the left leg freely but not with full vigor, moving it up and down restlessly and also drawing and pushing it downwards in response to efforts to call forth its use.

All the deep reflexes in the upper extremity of the right side were exaggerated as tested for the elbow and wrist jerks, and also for the knee jerks and the muscle jerks of the lower extremity. Foot clonus was marked and very persistent on the right. The Babinski sign was present, but elicited with difficulty, the plantar reflex response being so vigorous as to confuse the tarsal and the metatarso-phalangeal reflex. The Oppenheim and Gordon signs could be elicited.

Examining for touch, pain and temperature, the patient was moderately hypesthetic to pain and temperature in the right lower and upper extremities. Nothing could be made out as to touch. On account of the patient's mental condition and speech nothing as to topognosis or the senses of position and movement and stereognostic conception could be determined. All the reflexes and sensation were about normal in the left half of the body.

An ophthalmoscopic examination had been made by Dr. William K. Campbell, of Long Branch, who reported that it was difficult, owing to inability to get the patient to fix his eyes, to examine the discs carefully, but that the appearance showed some blurring but not a true optic neuritis.

It was noted that the head of the patient, who was held up and sat on the side of the bed during part of the examination, fell backwards, giving the appearance of a spasmodic retraction of the head. When lying down the appearance presented was also somewhat like this. Examination, however, showed that there was no spasticity of the muscles of the neck and that the head could be readily moved backwards and forwards and to either side by the hands of the examiner. After being thus moved it would again fall backward, the patient apparently not being able to hold it up. The tendency of the head to fall backward seemed to be due to loss of power in the muscles concerned in poising the head and in forward movements of the head.

An effort was made to determine whether or not hemianopsia existed, but without success.

The patient was reëxamined on Monday, July 30, 1906, by the writer, in consultation with the physicians and surgeons to the hospital, and Dr. T. H. Weisenburg. The examination revealed the conditions previously determined, and above noted, with some modifications and additions. In the first place, the paralysis of the leg, arm and face of the right side, especially of the leg and arm, was decidedly more profound than at the examination of three days previous. The patient could not be induced to make any movement of these limbs, nor could he be made to grasp the hand of the examiner. The tests for sensation were repeated with the result of making it more positive that hypesthesia to pain and temperature was present on the right. The observations as to ocular movements and the reflexes were practically the same.

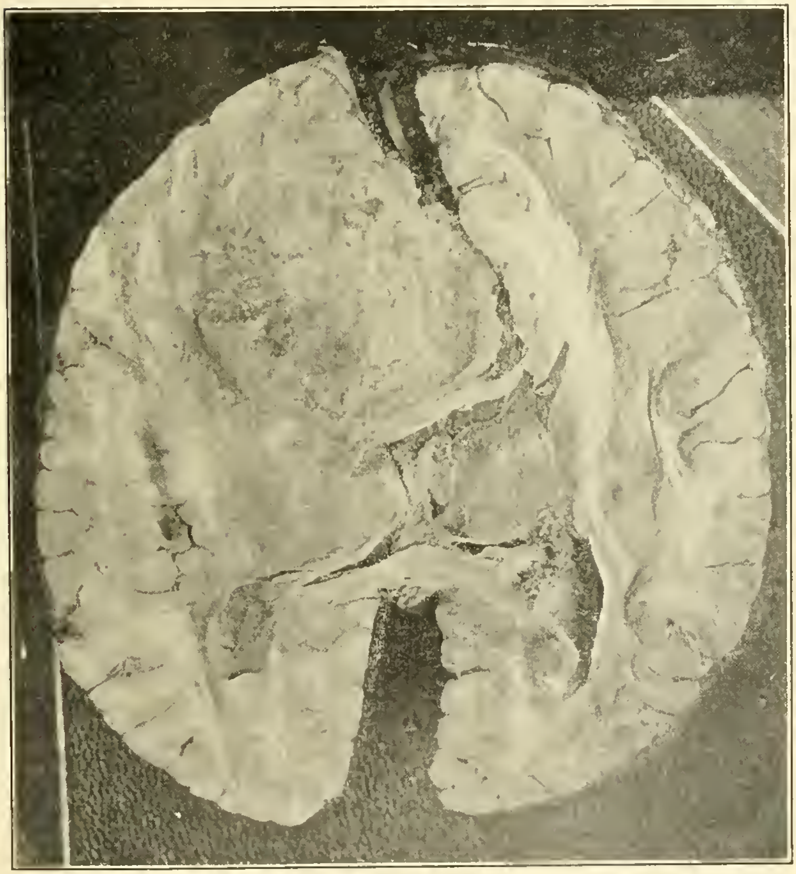
An ophthalmoscopic examination was made by both Dr. Campbell and Dr. Weisenburg. True choking of the discs was not present, but the discs showed blurring of their margins.

On one occasion, about a week before the examination, the patient had complained to his son about hearing some noises, asking the son whether he did not hear them. He apparently indicated the left ear by putting his hand to his ear, but as the right extremity was paretic this use of the left arm may have been due to impairment of power in the right. It was not quite clear whether or not these were real hallucinations.

As the result of the consultations it was decided to operate for decompression and exploratory purposes. Dr. Forman made a large osteoplastic flap, using the DeVilbus rongeur forceps, the opening being so planned as to be about two thirds in front and one third behind the central fissure. Its superior line was three and one fourth inches in length. The sides of the opening, about three inches in length, trended to a base line of about one and one half to two inches. Before and after the exposure of the dura the brain showed great tension as if a subcortical tumor might be present, but no growth was revealed by the exploration and it was deemed best to close the opening. The patient died within twenty-four hours after the surgical procedure.

At the necropsy which was performed by Dr. Weisenburg, the dura was somewhat adherent to the brain at the site of the operation. The superior, middle and inferior frontal convolutions were flattened and the fissures between them were almost obliterated. The orbital surface did not show any compression. Longitudinal sections of the brain made at various levels showed a large tumor which involved the interior of the left frontal lobe, compressing the right hemisphere as well as the left in a manner to be described. The tumor was soft in consistence, gelatinous in its structure, its border infiltrating into the surrounding brain tissue. It involved nearly the whole of the subcortex of the left frontal lobe. In its superior portion it was within one fourth of an inch of the cortex; anteriorly it was within one eighth to one fourth of an inch of the frontal tip; inferiorly it was within one half of an inch of the orbital surface, and laterally to within one half of an inch of the cortex. In the median portion in its widest area it projected to the right side, compressing the right frontal lobe, including the subcortex directly underneath the precentral convolution. Posteriorly the tumor compressed on the right side the caudate nucleus and to some extent the anterior limb of the internal capsule, but did not press upon the knee and posterior limb. It apparently here had made pressure upon some of the motor fibers coming from the precentral convolution. On the left side the tumor had compressed out of their proportionate relations the left caudate nucleus, the anterior limb of the internal capsule, the lenticula and the anterior portion of the thalamus.

The anterior limb of the internal capsule formed with the posterior limb a right angle and the posterior limb was considerably distorted. The tumor had obliterated the anterior horns of the lateral ventricles on both sides and extended as far back as the anterior portion of the third ventricle which was not compressed. The tumor had destroyed entirely the anterior and middle portions of the callosum. In its widest portion it measured three and one fourth inches in width and two and one half inches antero-posteriorly, although it was probable that it extended further by infiltration than was shown by the macroscopic examination. It was a sarcoma.



Photograph of a section through the upper part of the basal ganglia.

Sections made of the pons and peduncle showed a slight secondary degeneration of the pyramidal fibers of the left side.

The patient in this case had, in the first place, some of the well known symptoms of brain tumor, such as are usually present in a lesion of this description wherever situated. These were pain in the head, vertigo or dizziness, and a blurring of the optic discs which probably represented the beginning or the imminence of optic neuritis.

Among the most striking symptoms which seemed to be focal in character were the awkwardness or clumsiness of movement in the extremities of the right side of the body, which towards the termination of the case took the form of a hemiplegia; the weakness or paraparesis of both lower extremities, which was manifest before the right-sided hemiplegia became apparent; the aphonia or lowering of the voice; the tendency of the head to fall backwards, which was apparently due to a paresis or paralysis of some of the muscles concerned in holding the head erect, no spasticity being present; the involuntary evacuations from both bladder and bowels; and the mental apathy or feebleness. The deep reflexes in the right half of the body were exaggerated, and some hypesthesia to pain and temperature was present.

These symptoms are to be attributed in the first place to the large amount of destruction of the subcortex of the frontal lobe, and in the second place to the destruction of the knee and the anterior half of the callosum.

The hypesthesia to pain and temperature is probably to be referred to the involvement directly or by pressure of the subcortex of the gyrus fornicatus.

The exaggeration of the deep reflexes on the right side, including the Babinski sign and foot clonus, which were probably late symptoms, had for their pathological cause compression or direct implication of the pyramidal tract, the posterior extremity of the tumor having encroached somewhat upon the motor subcortex and having produced distortion of this portion of the brain and of the posterior limb of the internal capsule.

Such mental apathy, weakness, and disturbance as were present may be referred jointly to the involvement of the frontal lobe and the callosum. It is well known that psychic symptoms have often been observed in frontal and especially pre-frontal tumors, this being generally more marked when the neoplasm is situated in the left hemicerebrum. Mental symptoms have also been reported in some cases of callosal tumor.

The involuntary evacuations which were present for some days before the death of the patient furnished an interesting feature of the disease. In many cases of tumor of the brain the patients retain control over the bladder and bowels to a very late period, although this is not retained in other cases. The question of the position of the centers controlling these movements is interesting. Anal, vaginal and vesical centers have been placed in the paracentral lobule. The sphincters of the anus, bladder and vagina are doubtless controlled by cortical centers, the centers of one side being sufficient for this control. Destruction of the cortical centers of one side would not cause total paralysis of the sphincter muscles of the anus, bladder and vagina, whereas if these were destroyed on both sides this result would follow; and similarly it would be expected that destruction of the fibers going from these centers, or the commissural fibers connecting these centers through the callosum, would bring about this result.

To the writer the most interesting focal manifestation in this case was the tendency of the head to fall backwards, apparently because of paralysis or paresis of the muscles which hold the head in the erect position. This form of paralytic affection has, so far as I know, not been recorded. In another case recently observed and placed on record at the same meeting at which the present case was reported, a paralysis or extreme hypotonia of the muscles which support and poise the head was the feature of most interest, although the case in other particulars differed from that here presented and was probably an instance of cerebellar disease. In both of these cases no spasticity was present, but the reverse. The patient's head tended constantly to fall backwards, and apparently could not be supported in an erect position. Even when lying down this tendency was apparent and led to an examination being made for rigidity of the neck muscles, which however was absent; the head was easily movable in all directions by the hands of the observer, offering absolutely no resistance. It was clearly not a case of spasmodic retraction.

Whether the destruction of the callosal commissural fibers between the cerebral regions for the movements of the head and neck may not have had something to do with this symptom, is worthy of consideration. The head is carried erect and is poised in various positions through the interaction of a complicated bilateral musculature, all parts of which are probably governed largely by centers in each hemisphere. Destruction of these centers on one side produces therefore no appreciable effect; destruction of them or of their subcortical connections on both sides might produce a marked bilateral paralytic effect, and by the same reasoning, destruction of the callosal commissural tracts might produce inability to use these muscles.

Mott and Schäfer, as cited by the writer in his book on "The Nervous System and Its Diseases," "experimented with weak induction currents on the callosums of monkeys, and produced localized bilateral movements in all parts of the body. From before backward, in order, in different segments of the callosum, electrical stimulation caused movements of the head and eyes, the face, the shoulder, the trunk muscles, and the legs and tail. Cutting away one hemicerebrum, they found that by stimulating a thin middle strip of the callosum localized movements were produced on the side of the section, that is, on the side of the body opposite to that hemicerebrum with which the callosum was still connected, the order of movements being the same as above mentioned." As the result of these experiments, it would seem that a large destructive lesion on one side of the cerebrum, involving the anterior part of the callosum and the adjoining portion of the other hemisphere, would cause more or less bilateral paralysis of the parts controlled by the centers whose commissural connections were destroyed. These connections in the case here recorded were probably those of the centers concerned with movements of the head. These centers, it is well known, are situated well forward in the motor region, in such position that the tumor found in this case would be likely to destroy both their projection and commissural tracts.

The aphonia or apparent aphonia may have been merely a late symptom indicating great weakness, or may have been dependent somewhat on destruction of the commissural connections between the laryngeal centers. The patient also had apparently some aphasia, but this aphasia seemed to be more than it really was because of his loss or great impairment of vocalizing power. One, two and three days before death he responded clearly to questions asked him by "yes" or "no," or made use of single words, or even short sentences, but it was always necessary to have the ear of the listener close to the face of the patient in order to understand what he said. The following are some illustrations of conversation held in this way: When a watch was held before him, after looking at it intently, he said "watch" in a whisper or very low tone; other objects, as a pencil and key, immediately afterwards presented to him, he called also a watch in the same tone.

Several days before his death the patient became unable to stand or walk without support, this apparently being due to great clumsiness and weakness of the right half of the body, which became a hemiplegia shortly before death, and also perhaps to some extent to the much less degree of weakness of the left side. A species of paraparesis has been observed in some cases of callosal tumor. The hemiparesis or hemiataxia, or clumsiness of the limbs on the right side was doubtless due to the prefrontal and midfrontal disease, the late hemiplegia resulting from pressure and direct encroachment on the motor region.

I have reported this case as one of somewhat unusual character in that it presented at least two symptoms unique or almost unique, as well as other well-known manifestations of frontal, subcortical, and callosal tumors. The literature of callosal disease has recently been well presented by Bruns, who, including his own, collected twenty-six cases in which this structure was involved. In referring the clinical phenomena to the lesion found, most stress should of course be laid upon the frontal subcortical growth, this being very large, although the involvement of the callosum must also have recognition.

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A FURTHER CONTRIBUTION TO PALLIATIVE OPERATIONS FOR BRAIN TUMOR.*

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Since the publication of the paper by Dr. Charles H. Frazier and myself,[1] some articles of much interest bearing on decompressive operations have appeared. It is the author's intention to refer briefly to the more important of these, to report some cases not included in the former paper with Dr. Frazier, to discuss palliative operation where choked disc is the only important sign of intracranial disease, and to report cases of this character, to speak of rapid increase of blindness under certain conditions as a result of decompression, and of palliative operation performed when the respiration has ceased, but the heart is continuing to beat; and to give the present condition of some of the patients described in the previous paper.

Recurrence of choked discs may occur after decompression, as described by de Schweinitz and Thomson.[2] Their patient was trephined for the relief of the symptoms of a tumor, believed to be situated in the right motor area. The tumor was not removed. Three and one-half months after operation the double optic neuritis had subsided and there was postneuritic atrophy. One year later the patient returned with all the symptoms of brain tumor re-established, from which he had been entirely free, totally blind and with double optic neuritis, the apex of each swelling being 6 D., or as high as it had been before the original operation.

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[1] The Journal A. M. A., Sept. 1, 1906, p. 679.

[2] Arch. Ophth., 1905, xxiv.

De Schweinitz[3] states that his experience has been somewhat as follows: Usually there is no marked change in the swollen nerve head after trephining in which the dura has been opened, or after the removal of a tumor, until the tenth to the fourteenth day. He has not seen decided subsidence of engorgement edema at the end of forty-eight hours, except in one instance, when within that period, following the removal of a cerebellar cyst, there was a decline of several diopters in the height of the swollen papilla. It is not uncommon to find within the first thirty-six hours, and even earlier, a diminution in the congestion of the retinal vessels. After the tenth to the fourteenth day there is gradual subsidence of the neuritis, from six weeks to two months being required for its complete disappearance. The ultimate vision may be (a) better than that which existed prior to the operation: (b) equal to that which existed prior to the operation; (c) worse than that which existed prior to the operation. Sometimes total blindness results.

In the cases with preservation of vision, either as good as or better than that which the patient possessed before the operation, the sight may be better in one eye than in the other, or exceedingly defective in one eye and good in the other, or one eye may be blind and the other retain or regain excellent vision. The most satisfactory results follow the reduction of great intracranial pressure, provided the papillitis or choked disc has not so long existed that it has already destroyed the optic nerve fibers. He gives the results in three cases.

In a certain number of cases de Schweinitz found that during the first day or two after trephining there may be a slight increase in the neuritis, or a slight increase in edema, associated with fresh hemorrhages. This is apparently of no importance, as the added edema and fresh hemorrhages disappear in the subsequent general subsidence of the swelling. In some of these cases there are marked degenerative changes in the retinal vessels. De Schweinitz, on several occasions, noticed a temporary depreciation of vision within the first week after the operation, probably due to shock or hemorrhage, and which, apparently, is of no importance in the subsequent preservation of vision, provided the primary vision has been good and the neuritis of comparatively short duration. Where little vision exists prior to trephining, this may rapidly disappear after trephining, especially if there has been much hemorrhage.

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[3] Univ. Penn. Med. Bull., April and May, 1906, and personal communication.

Bruns,[4] in his excellent work on tumors of the nervous system, puts the matter of decompression in this manner: "Shall one refuse this possibility [palliative operation] of considerable relief from the disorder to those in whom a localizing diagnosis is not possible even after longer observation, or removal is unattainable? I think we are not justified in so doing." In speaking of saving sight by decompression, Bruns mentions that many persons with brain tumor may live long, and that all the symptoms of tumor may disappear spontaneously, except that blindness may become complete. Such a case seems to be Case 5 of the paper by Dr. Frazier and myself, which is referred to further on in this article. In cases of this character, decompression would probably have saved the sight.

The danger of blindness is great when vision begins to fail with choked discs, and flecks of fatty degeneration in the retina are always serious. If the tumor implicates the basal visual tracts, decompression can not prevent blindness. Usually, this may be recognized by typical contraction of the visual field, and then decompression is not to be recommended.

Some have held that it would be well to rest content with decompression when the tumor is in the posterior cranial fossa, and not to attempt its removal because of the danger in so doing; this opinion Bruns does not share. I must confess that I am partly persuaded that in many cases decompression is the best procedure, as the attempt to remove a tumor of the posterior cranial fossa usually terminates fatally, or the condition of the patient after the removal of a tumor from the cerebellopontile angle is so pitiable that death is welcomed as a release. It is true that a few brilliant cases are on record, but many failures are recorded. Further improvement in technic may make these operations on the posterior cranial fossa more successful. I am referring especially to the cases in which the tumor is in the cerebello-pontile angle.

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[4] Die Geschwülste des Nervensystems, 2d edition, p. 282.

Relief of pressure is not obtained in all cases by trephining and opening the dura to allow escape of fluid. and the decompression is not without danger. Bruns advises against it when the patient is in deep stupor, and, at the same time, the symptoms indicate that the tumor is very large; palliative operation could only serve to restore a consciousness which mercifully has been lost. He is not referring to decompression in the sudden attacks of syncope in cases of brain tumor; such as I have seen repeatedly. Bruns thinks the dura must be opened in palliative operations, and this view I fully share. In one of his cases. Bruns recommended operation over the left parietal lobe, the prolapse of the brain that followed caused ataxia and optic aphasia, so that under some conditions the results of decompression may be serious. A less important region should be chosen for decompression.

When the cerebrospinal fluid does not escape after trephining, and relief of pressure is not obtained, Bruns recommends puncture of the ventricle, following the teaching of Wernicke, Sahli, Kocher, and von Bergmann. Continuous drainage may be employed. Lumbar puncture Bruns regards as dangerous in brain tumor, as the medulla oblongata may be crowded into the foramen magnum and obstruct it so as to prevent the escape of fluid. This opinion experience has fully confirmed.

It is necessary to mention that some authors speak of trephining as synonymous with decompression. The opening in the skull should be larger than that made by a trephine when relief of pressure is sought.

Alfred Saenger[5] reports 19 cases of brain tumor with palliative operation. In two cases, improvement occurred after the trephine opening was enlarged so that more cerebrospinal fluid could escape. In two cases trephining had no beneficial results as the tumor was too large. The condition was made worse only in one case, and in this the tumor was at the base. In all the other cases beneficial results were evident. Saenger states that Finkh, in 1904. collected 31 cases from the literature; in 30 of these the choked disc disappeared; in one case the symptoms increased. According to his

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5. Klin. Monatsbl. f Augenheilk., 1907, p. 145.

statistics, palliative operation causes improvement of vision in 73 per cent. of the cases.

Saenger chooses as the time for operation the commencement of diminished visual acuity. He selects the region where the tumor is supposed to be, but when this is impracticable he recommends the decompression to be done over the right parietal region.

Lumbar puncture, or puncture of the lateral ventricle, is not to be compared with decompression, as either method is much less effective than decompression.

The dura was always opened in von Krüdener's[6] cases. Only those patients were operated on who were in danger of complete blindness and were in a late stage, otherwise the author thinks the results probably would have been better. He states as certain that trephining causes altered conditions of the circulation and brings about a reduction of the choked discs, which is noticed in the first twenty-four hours following a diminution in the congestion of the blood vessels. The diminution in the swelling begins, however, on the second or third day, and the swelling does not completely disappear after two or three weeks, as the papillæ are still somewhat elevated. If the trephine opening grows together in such a way as to cause a continuance of the intracranial pressure, the papillæ soon attain their former prominence.

He does not hold a very positive opinion regarding the effect of trephining on inflammation of the optic nerve, as he had only one case in which probably basal meningitis was present, although the result seemed to be good. He has not observed a sudden reduction in the papilla immediately after the operation, although he has observed the eyegrounds during and immediately after the opening of the cranium. The change was always very gradual, and not until the evening following the operation or the next day could he say positively that the veins were somewhat smaller and the papilla and adjacent retina somewhat paler, although the arteries were not wider. Changes in the hemorrhages, in degenerative foci in the macula, or in the edema of the retina when present were always slight.

Subjective disturbance, unconsciousness, especially from brain tumor, disappear at times entirely, and he thinks trephining is proper when pain and the feeling as though the head would burst are present and blindness threatens, although he believes that a tumor grows more rapidly after the cranium is opened. The pressure of the distended third ventricle and of the distended floor of this ventricle on the chiasm can be removed at times by trephining. After we learn to distinguish between increased intracranial pressure of inflammatory origin and that of non-inflammatory origin, trephining will be more advantageous. There is more danger of prolapse of the brain with hydrocephalus in children. In hydrocephalus of childhood he found that the protruding brain so closed the opening in the skull in some instances that no liquid escaped and the bandages remained dry; in other cases the bandages were soaked the day following the operation.

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[6] v. Graefe's Arch., 1907, lxv, 69.

Single or repeated lumbar puncture in twelve cases of brain tumor produced no effect on the choked discs. Also in cerebral tuberculosis lumbar puncture produced no improvement in the condition of the eyegrounds. Division of the dura of the optic nerve, recommended by de Wecker, was useless, and some stretching of the nerve occurred because of the small field of operation. In a case of cerebral tuberculous meningitis, in which an exudate was found in the sheaths of the optic nerves, lumbar puncture produced no visible change in the eyegrounds. Lumbar puncture has been believed by quite a large number of writers to aid in recovery from meningitis, and where recovery occurs the optic nerves may be benefited, and puncture should be employed in syphilitic meningitis with papillitis. The effect is not to be determined by the ophthalmoscope, and consists probably in slight relief of pressure from the papillæ and brain, so that more blood may reach the diseased parts.

von Krüdener observed in one case that after removal of the brain pressure meningitis developed on the brain and optic nerves, and notwithstanding the intense inflammation the choked disc gradually and constantly diminished. This would seem to me to indicate that decompression may be useful in inflammatory conditions, but in a case of purulent meningitis, that Dr. Frazier and I recently have had, palliative operation was followed by death within a few days. von Krüdener and others (Bruns) have observed that after trephining in hydrocephalus choked disc disappeared, although no liquid escaped on account of the prolapse of the brain.

It is not necessary to assume the existence of phlogogenous substances in the cerebrospinal fluid to explain the choked disc of brain tumor and of hydrocephalus, and the inflammatory appearances so often present are a result of stagnation of lymph and of edema. In local brain pressure von Krüdener accepts the opinion of von Graefe, and explains them as the result of compression of the cavernous sinus and congestion in its venous tributaries. He thinks the future must decide whether it would be advisable to remove the bone about the chiasm under certain conditions to relieve choked discs. He has done puncture of the ventricle twice with the drill, according to Kocher's idea; the cases were not very suitable and the choking was not affected thereby. Wilbrand and Saenger[7] believe that when the pressure is lowered in the spinal canal the medulla oblongata is pressed downward, and until the pressure is restored the circulation of fluid from the cranium to the spinal column is prevented.

Lumbar puncture has usually little effect on choked disc for several reasons, the pressure is not always the same in the vertebral cavity and the cranial cavity, and probably is higher in the cerebral ventricles than in the sheath of the optic nerve. After lumbar puncture the pressure on the brain is relieved and the circulation of blood is improved, and thereby poisonous products are removed; aud probably it is useful in syphilitic choked disc. Lumbar puncture has been recommended by many as a therapeutic and diagnostic means for syphilitic choked disc.

von Krüdener speaks of two other forms of choked discs, viz., from aneurism of the carotid artery and after operations on the ear. The former may be unilateral one or two years, and later become bilateral, and may be accompanied by very high swelling. The latter occurs with abducens paralysis. Gradenigo assumes that a meningitis occurs at the point of the petrous portion of the temporal bone and extends to the optic nerve; other authors speak of edema and compression of the wall of the sinus from an edematous brain.

Risien Russell[8] recognizes that it is useless to trephine for the relief of intracranial pressure when the patient is deeply comatose or the heart has failed, but he thinks it should always be attempted when the respiration alone has ceased and the heart is continuing to beat, even though the chances of saving the patient's life may be remote. He believes that some reported cases seem to show that by removal of a part of a tumor the growth of what is left may be retarded. This has always seemed to me very doubtful. He says he is strongly impressed with the value of trephining for the relief of optic neuritis in order to preserve sight, that he recommends that no patient suffering from intracranial tumor should be allowed to become blind when in this operative measure we have a means of preventing it, provided the operation is undertaken early enough. He does not believe that trephining will relieve every form of optic neuritis, whether due to increase of intracranial pressure or not. It would be worse than useless, he thinks, to trephine when the optic neuritis is of toxic origin. It seems to me, however, that this question is open to discussion.

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[7] Die Neurologie des Auges, iii.

[8] Brit. Med. Jour., Oct. 27, 1907.

Lumbar puncture can only be regarded as a temporary measure and can not be expected to relieve symptoms in the permanent way that trephining does. In lumbar puncture he thinks we have a means of relieving urgent symptoms of pressure, when to wait for a surgeon to trephine would be to allow the patient to die; or it is valuable when the patient's condition is too serious to permit the major operation, even when a surgeon is available. In both these classes of cases relief by lumbar puncture may be sufficient to permit of the major operation of trephining later. Lumbar puncture, in his opinion, is valuable for diagnosis and for the relief of urgent symptoms in cases of intracranial tumor, but is not likely to permanently relieve symptoms and prolong life or to effect a cure.

J. Mitchell Clarke[8] recommends that when no localizing signs are present the trephining should be done at the site of a persisting tender area, and in one of his cases a large tumor of the upper parietal lobe was found in this way. Dependence on a tender area in one of my cases, Case 2 of the paper by Dr. Frazier and myself, proved unreliable. Clarke thinks a palliative trephining makes a later diagnosis of location more difficult.

Mr. Marcus Gunn[8] states that when the papilla becomes more opaque and sometimes more prominent, the hemorrhages increase in size and number, there are inflammatory exudations on the disc and surrounding retina and vision has become impaired; the prognosis regarding the saving of vision is bad after relief of intracranial tension, although he has known useful vision obtained even in such unfavorable circumstances. Palliative operation to save vision is useless in the stage of gradually decreasing vascularity of the papilla, when parts of its surface become even paler than normal, while the prominence either persists or slowly subsides and the branches of the central artery become diminished in breadth.

Certain conditions Mr. Gunn emphasizes as important, viz.: The degree of swelling of the papilla in itself is not an accurate indication of the visual prognosis, and all the ophthalmoscopic appearances must be considered. The presence of retinal hemorrhages does not contraindicate surgical intervention, nor is retention of normal vision before operation a safe guide, though it is more favorable. Vision previously normal may be lost after operation. Visual improvement may ultimately be very satisfactory, even though long delayed. In one of his cases very slight improvement in vision was observed after two months, yet two months later the result was most satisfactory, one eye having regained 6/5 from 6/18, and the other 6/6 partly, from mere hand reflex. Some of the most striking results in vision he has observed after operation were in cases of cerebellar tumor. So far as vision is concerned he has found that it makes little difference whether the tumor is removed or whether the dura is merely freely opened.

Mr. Gunn has had no experience regarding the relief of tumor papillitis by lumbar puncture; he believes it would not afford so thorough a relief as the cranial operation, and would need to be frequently repeated. He does not favor opening the optic nerve sheath immediately behind the globe. We may well, I think, ask why it should be recommended, as it does not relieve the other distressing symptoms. It would have to be performed on both optic nerves, and the opening is liable to soon close up.

Mr. Gunn mentions that tumor papillitis is commonly associated with hypermetropia and is relatively rare in myopia. It appears as if in myopia an increased pressure within the sheath space is less likely to affect the optic nerve and more likely to be relieved by filtration or absorption of the excess of fluid.

H. C. Thomson[9] reports a case of tumor supposed to be in the temporal lobe in which palliative trephining caused gradual subsidence of the optic neuritis, and in a few weeks the discs showed only an insignificant trace of infiltration. During the six months following the operation the symptoms did not increase, but later did so. He refers to a case of cerebellar tumor in which palliative operation also was effective.

Merz[10] has studied the causation of optic neuritis. He states that in his experiments, in which he produced increased intracranial pressure by injecting fluid, changes in the rabbit. consisting of venous hyperemia and arterial anemia of the retina and the appearance of vessels in and about the disc which usually are not visible in the healthy eye, can be brought about almost instantly. In dogs a longer time is required for the development of the venous hyperemia; the difference is owing to the different arrangement of the vessels in the eyes, that in the dog more nearly resembles the condition in man. The further changes in the fundus of the dog appear two or three hours after the beginning of the increase in tension. The papilla becomes gradually edematous, loses its sharp outlines, projects forward and acquires a rosy hue. After eight or ten hours the arteries and veins arch perceptibly in passing from the papilla to the retina, and they appear blurred, veiled or even in places interrupted from the edema and cloudiness of the tissues in which they run. Further observation is usually impossible because the animal dies.

In every case, dog or rabbit, the subvaginal space about the optic nerve was found to be dilated. The cerebrospinal fluid passes between the dural and pial sheaths to the bulbar end of the space which ends in a blind sac. Since the intracranial tension does not diminish the liquid distends the subvaginal space, and more excessively at its bulbar end. Thus arises the ampulla-formed dilatation described by Manz, Schultén and others. Frequently a round-cell infiltration is found in the sheaths of the nerve and the surrounding connective tissue and the subvaginal space suggesting a perineuritis.

No one doubts the possibility of a choked disc arising in certain cases after the introduction of various poisons into the body. Experimental and clinical observations teach us that undoubtedly choked disc develops at times in consequence of local irritation of the papilla and optic nerve from toxins and various poisons. Solowieff found that the introduction of the *Staphylococcus aureus* into the vitreous was in most cases followed by papillitis and choked disc. Selenkowski made similar observations. Hallermann saw choked disc in a case of erysipelas of the face, Adamück in a case of retention of urine. The inflammation theory is admissible for some cases of choked disc but not for the majority of cases. No one has determined the presence of phlogogenous substances in cases of brain tumor, and it is in these cases that choked disc most frequently appears. For the unprejudiced investigator the mechanical theory has more points of plausibility than the inflammatory. Twenty hours of slightly increased intracranial tension is sufficient in dogs to cause edema of the optic nerve.

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[9] Brit. Med. Jour., Dec. 21, 1907, p. 1761.

[10] Arch. Ophth., 1901, xxx, p. 349.

White blood corpuscles emigrate in large numbers from the vessels and form the round cell infiltrations, especially in the parts most exposed to pressure, such as the outer sheath and the neighboring tissues. Thus increased intracranial tension suffices to cause symptoms of stasis and even of inflammation in the optic nerve and papilla.

Increased intracranial tension alone is sufficient to produce choked disc. There is similarity in anatomic construction between the eye of the dog and that of man.

Brudenell Carter, in three cases of choked disc, made an incision through the outer sheath of the optic nerve, and in each case vision improved and the symptoms of papillitis decreased and all the other symptoms of increased intracranial tension vanished.

Other important papers on cerebral decompression that have appeared since our former paper[1] are those by Starr,[11] Chance[12] and Stieren.[13]

It is sometimes extremely difficult to decide what is best to be done when choked discs are the only sign of intracranial disease. The patient may otherwise appear to be in perfect health, and the physician hesitates to recommend the opening of the skull. It may be difficult to convince the patient and his relatives that a neoplasm probably is present, especially if the physician also be in some doubt about the diagnosis, and he is likely to be if choked disc is the only sign. There is the possibility that the swelling may be caused by nephritis or meningitis, and as yet we are uncertain whether or not the choked disc of meningitis is improved by decompression.

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[11] THE JOURNAL A. M. A., Sept. 22, 1906, p. 926.
[12] Penn. Med. Jour., August, 1907, p. 877.
[13] Ophth. Rec., March, 1908, p. 139.

Antisyphilitic treatment may be beneficial and cause great improvement for a time, as in Case 1, but such treatment is known to be useful occasionally in glioma or other form of cerebral neoplasm. Valuable time may be lost by attempting to relieve the symptoms by mercury and iodid, and yet a too hasty operation is to be deprecated. The suggestion of decompression seems so terrifying to the patient, and so unnecessary in view of what appear to him as insufficient symptoms, that he is likely to be driven to consult some other physician, and possibly he may be assured in this consultation that he need have no fear and that recovery without operation will occur. When the patient finally consents to decompressive operation, valuable time often has been lost, and it may be too late to save the eyesight. The following case, in which operation was unavoidably delayed, illustrates these statements:

Case 1.—Miss K., aged 20 years, was referred to me Nov. 15, 1906, by Drs. Ferguson and Schneiderman.

Examination.—Dr. Schneiderman first examined her eyes Sept. 29, 1906, simply because she complained of headache. He found slight optic neuritis and a flame-shaped hemorrhage in each eye, more marked in the right eye. An increase of optic neuritis was observed October 6, and by November 15 the swelling was 4 or 5 D. in each eye, and more in the right eye. Central vision was full, but the fields were contracted. Headache had been severe since June, and was both frontal and occipital. She had not had vertigo, but she felt as though she staggered, and yet her mother had not seen any ataxia. Nausea was felt November 14. Menses were always late, and were more delayed during the past few months. She had complained of numbness occasionally for more than a year, from the back of the head, down the neck into the left shoulder, and down the left arm. It was a sensation of pressure, not of pain. She had not had convulsions. Dr. Schneiderman found the pupils normal and no paralysis of ocular muscles.

In my examination the left corner of the mouth did not seem to be drawn up quite so well as the right, possibly the left lids were not closed so forcibly. The right patellar reflex was weak, the left was uncertain. There may have been a slight Romberg's sign, but it was not positive. In walking she had the sensation of going toward the right, but she did not stagger. The Achilles reflexes were about normal. The urine showed only a trace of albumin and no casts.

Dr. de Schweinitz reported Nov. 22, 1906: Central visual acuity normal. Visual fields and accommodative power normal. No diplopia and no paresis of any external ocular muscle. Double choked discs, with many hemorrhages, the elevation of the discs being 5 D. The ocular symptoms, he thought, were those of brain tumor.

Treatment.—Syphilis seemed very improbable, but my advice was to try antisyphilitic treatment, pushed rapidly, with careful observation of the eyegrounds, and with the understanding that a palliative operation should be performed if any further destructive changes were observed. Decompression would not have been considered at that time by the patient or her relatives, but I warned them earnestly of the danger of blindness they were incurring in delaying it. I did not see the patient again until June 15, 1907. She had improved under iodid and mercury. The headache had disappeared. Dr. Schneiderman found the fields better, the contraction was slight, vision was normal, and the swelling of the discs was slight, not over 1 or 2 D. The left disc was a little pale. The patellar reflex was present on each side, but was diminished.

She consulted me again Oct. 5, 1907. She had disobeyed instructions and had not visited Dr. Schneiderman for three months, and her eyes were worse. She had now about 6 D. of swelling, more in the left eye. She had not taken mercury and iodid properly. Vision was 5/4 in the right eye and 5/5 in the left. She had had much vertigo, and at times lost her sight temporarily. My examination failed to reveal other important symptoms. It seemed too risky to delay decompression by attempting further treatment with mercury and iodid, and I advised her to submit to operation. She was unwilling to do so, and consulted Dr. Van Pelt, who later referred her to Dr. Mills, from whom she received the same advice which now she accepted. I am indebted to Dr. Mills for the notes taken after the patient came under his care.

Dr. de Schweinitz made an examination of the eyes Jan. 14, 1908, and reported as follows: The vision of the right eye is reduced to the ability to distinguish hand movements; of the left eye the largest letter on the type card, that is, Snellen 200, could be distinguished uncertainly at about 2 feet. Both discs showed the decided changes of beginning postpapillitic atrophy. The right disc is swollen still about 3 or 3.50 D., the left disc somewhat higher, between 4 and 5 D. It was difficult to make these measurements exactly as she was lying in bed.

Dr. C. H. Frazier performed a palliative operation Jan. 10, 1908.

The report of Dr. Van Pelt, who has had the patient under observation since the operation, is as follows: Jan. 30, 1908. In the right eye the swelling of the optic disc has decreased from 5 to 2½ D. since the operation. In the left eye the swelling has decreased from 3½ to 2 D. The optic nerve shows evidence of increasing atrophy, and the retinal arteries are becoming more attenuated. These atrophic changes are more decided in the left eye. The vision has markedly decreased in each eye in the last ten days.

Case 2.—*History.*—Miss F., 19 years of age, was referred to me from the service of Dr. John H. Musser, Sept. 5, 1907. She had had headache since April, 1907, nearly every morning. The pain was back of the right ear and was sharp. This area of the head was not tender to touch. The headache disappeared almost every day about 11 o'clock, although occasionally it persisted all day. It was more severe in April or May, 1907, and at that time she had much nausea. She vomited often when she had the pain, and sometimes when the pain was not severe. She had some diplopia in looking toward the right, but she thinks it was formerly in looking toward the left. She was of fragile build, weighed 85 pounds, and appeared anemic. The patient's mother died of tuberculosis, though she herself had never had any distinct pulmonary trouble, and yet Dr. A. Fife found some suspicious signs at the right apex, but he was not positive of their value.

Examination.—A report of Dr. Fife from Dr. Musser's office stated that the urinary examination was practically negative, but the specific gravity was low, 1007, with the faintest possible trace of albumin. The microscopic examination was negative. There was no indican, no urobilin and no sugar.

Dr. T. B. Holloway reported Aug. 21, 1907:

Vision, O. D., with plus .75 ax. 90 (now wearing) = 5/12.

Vision, O. S., with plus .75 ax. 90 (now wearing) = 5/12.

Pupils are 7 mm. in diameter, each reacts slightly to light, accommodation and convergence. When eyes are rotated laterally to the right and to the left a nystagmoid movement develops at the outer limits of rotation, probably slightly more marked on the right side. There is a slight limitation of outward rotation of the right eye, questionable on the left. The diplopia fields show a paresis of the right external rectus. In each eye the media are clear and the fundus shows a choked disc, the height of the swelling being 4 to 4.50 D. There is no demonstrable ocular lesion of tuberculosis.

Sept. 2, 1907. Fundus condition unchanged. Fields show moderate contraction. Direct light reflex practically abolished in the left eye; with condensed light a feeble attempt at iris contraction can be noted at the lower pupillary margin. I examined this patient repeatedly at intervals of several weeks, but was unable to find any other signs of organic disease.

Dr. de Schweinitz wrote on Sept. 23, 1907: "She has very decided choked discs, a little greater on the right than on the left side, and somewhat greater now than when you received the report from Dr. Holloway on August 15. There is no hemianopsia, but there is a paresis of the right external rectus."

An examination of the blood gave: hemoglobin, 72 per cent.; red blood corpuscles, 4,960,000; white blood corpuscles, 10,000. Differential blood count: polymorphonuclears, 66 per cent.; large mononuclears, 5 per cent.; lymphocytes, 25 per cent.; transitional, 2 per cent.; eosinophiles, 2 per cent.

It seemed to Drs. Musser, de Schweinitz and Frazier and to me advisable to have a decompressive operation in order to save vision. Dr. de Schweinitz wrote Nov. 20, 1907: "I examined Miss F. yesterday and found the vision very much reduced as compared with what it was when Dr. Holloway first examined her on August 15, when it was 5/12, while to-day it is only 5/30, and that not quite perfectly. There has been very little change in the appearance of the optic nerves. They are swollen moderately, not more than 2 or 2½ D., possibly 3, while at that time they were swollen to 4 or 4½ D. Somehow the discs do not impress me as very surely dependent on increased intracranial pressure. I presume, however, that a simple trephining could do no harm, although I should think it very desirable to make it as nearly a bloodless operation as possible, because a very little additional drain would surely very greatly depreciate the vision. The results of trephining, however, I am inclined to think are very problematical." This latter view we all shared.

Operation.—A decompressive operation was done by Dr. C. H. Frazier Dec. 3, 1907. A small hernia formed at the site of operation. It was painful to touch.

Dr. de Schweinitz reported December 10. There is absolutely no change, the neuritis being exactly of the same height that it was before the trephining and the visual acuity also is unchanged. December 23. There is marked improvement in the vision of the left eye, with stationary improvement in the neuritis, possibly a little subsidence; stationary vision in the right eye with marked increase in the swelling of the disc.

Notes made by me Jan. 14, 1908, stated that the patient had been in the hospital about a week after the operation, and during that time and the week following the return to her home she was entirely free from headache. When the headache returned it was not so severe as it had been before the operation. She had had very severe headache during the past two or three weeks. A careful examination failed to reveal any localizing signs of disease. Lumbar puncture was performed by Dr. Frazier; this was followed by severe headache, a flush over head and neck, feeble respiration, and very slow pulse; from this condition she soon rallied. She was discharged, January 22, in much better condition.

In the following case the question of decompression was considered, but the operation was not performed:

CASE 3.—C., a boy, aged 14 years, was sent to me Dec. 19, 1907, by Dr. G. E. de Schweinitz, with the following report: Double optic neuritis is subsiding, with every indication of rapid atrophy beginning. The swelling of the disc is now about 4 D.; apparently no paralysis of any external ocular muscles exists; very curious symmetrical defects are seen in the nasal field of each eye. Such defects, however, I think are now universally conceded to be due, not to lesion around the chiasm, but to lesion of the optic nerve itself, and I have seen them before with optic neuritis when there evidently was no chiasm lesion.

History.—The boy had scarlet fever at the age of 3 years, which left a discharging ear. In March, 1904, he was operated on for mastoiditis. He had very severe nephritis at that time, and was in a serious condition after the operation. The discharge of the left ear continued after the operation. Optic neuritis was discovered in October, 1906. He had not had headache, dizziness, nausea or vomiting.

Examination.—I found that the left pupil was a little larger than the right, both irides responded to light, the left iris not so promptly as the right. He was partially deaf in the left ear. A careful examination failed to reveal other signs of disease. The case seemed to Dr. de Schweinitz and to me unsuitable for a palliative operation. A cerebral abscess might be present, and if this were the cause of the optic neuritis decompression might be of benefit. Rapid optic atrophy was present, and the report of the urinary examination was: one-fifth of 1 per cent. of albumin; no sugar; hyaline casts and cylindroids; and a specific gravity of 1016. It seemed probable, in the absence of all other cerebral symptoms, that nephritis was the cause of the atrophy, and a palliative operation was not performed.

The increase in the blindness which follows relief of intense intracranial pressure, and possibly the hemorrhage into the retina which occurs in some instances, as in Cases 4 and 5, may be explained by the sudden relief of the extracerebral pressure, with persisting intradural pressure. Cushing[14] remarks: "It must be remembered, however, that the sudden removal of pressure from the brain when the blood pressure has been forced to considerable heights, may be followed by a paralysis instead of a release from the major compression symptoms. The occasion of this is readily brought out by postmortem examinations, which, under such circumstances, oftentimes discloses a brain and medulla of a uniform cherry-red color, from the widespread extravasation of blood, due to the multiple rupture of the minute blood vessels. The external supporting pressure of the high intracranial tension has been suddenly removed, leaving the internal or intravascular pressure too great for the strength of the vessel walls."

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[14] Am. Jour. Med. Sciences, September, 1902.

De Schweinitz thinks the increase in blindness which occurs in advanced cases is caused by the loss of blood in the operation (personal communication).

The following two cases illustrate the occasional occurrence of retinal hemorrhage after palliative operation:

Case 4.—This was a case of cerebellar cyst, in the service of Dr. Wharton Sinkler. Dr. de Schweinitz stated that there was exceedingly rapid subsidence of the optic neuritis, but, in spite of it, rapid loss of vision occurred. There were symptoms of cerebellar tumor, and the operation, in two stages, was performed by Dr. Wm. J. Taylor. A cyst protruded when the skull was opened on the left side, ruptured spontaneously, and a stream of clear, straw-colored fluid squirted at least a yard. Considerable hemorrhage occurred at the first operation. The boy improved after the operation, except that immediately following it a hemorrhage developed in the right eye, and resulted in complete loss of vision.

Examination.—The notes as obtained from Dr. de Schweinitz and Dr. Holloway are as follows:

Oct. 1, 1907.—O. D., one letter on 6/30, one letter on 6/22; Snellen. O. S., 4 letters on 6/6. Snellen. Pupils 4½ mm., react to direct and indirect light, accommodation and convergence. Media clear; each eye shows a pronounced choked disc, height 6 to 7 D. One rather marked hemorrhage at the lower edge of swelling in O. S. No macular stellate figure. Convergence exceedingly poor, O. S. usually deviating. No hemianopsia. Will attempt fields later. T. B. H., fields taken October 4.

Oct. 8, 1907.—Optic neuritis (choked disc) O. D. + 8 D.; O. S. + 7 D. deS.

October 18.—O. D., height of swelling about 7 D.

October 25.—O. D., no change. O. S. shows beginning stellate figure in macula. T. B. H.

November 5.—O. D., disc + 3-4 D.; O. S., + 5-6 D. deS.

November 10.—Vision, O. D., doubtful light perception. O. S., 4/20 and one letter on 4/15. O. D., disc 3-4 D.; O. S., 4-5 D. Vessels in each eye 1-1½ D. higher. deS.

November 15.—O. D., blind; O. S., 5/15; O. D., no light reflex, in O. S. prompt. Indirect light reflex absent in O. S., present in O. D. Both react to convergence and accommodation. O. D., 4 D.; O. S., 5 D. T. B. H.

November 19.—Disc swelling about 3. Edges of disc beginning to appear. deS.

December 3.—O. D., + 4 D.; O. S., + 5 D. deS.

December 6.—O. D., blind; O. S. = 6/30. Fundus as when last seen. T. B. H.

In the macular region is a faint delicate, almost complete stellate figure, the striæ showing as milky lines rather than pure white. Within this area are several short white striations. At the outer limits of the stellate figure are several linear hemorrhages. One fresh hemorrhage on temporal slope of swelling. O. S. still some evidence of hemorrhage below disc. Height of swelling 7 D. In the macular region a number of fine yellowish spots, associated with several punctate hemorrhages. (T. B. H.)

Jan. 10, 1908.—Seen in out-patient department. Discharged from house Dec. 31, 1907. O. D. blind; O. S. 6/60. O. D. media clear, disc shows picture of subsiding neuritis, outline of disc distinctly seen, pale, edges blurred, arteries contracted, moderate perivasculitis; granular macula. O. S. media clear. fundus as in O. D. except in immediate region about disc, especially on temporal side, are a few punctate exudates. No hemorrhages in either eye. Height of filled-in discs, 2.50 D.

Case 5.—W. R., 43 years of age, white, house painter, was a patient of Dr. C. S. Potts, with whom I saw the patient, and to whom I am indebted for the notes.

History.—Family history negative. He has used considerable alcohol, has had much family trouble of late. Syphilis denied. He was struck in the right temporal region with brass knuckles one year before admission (Nov. 6, 1907). Symptoms of present trouble began about September, 1906, with headache and dizziness. On admission he complained of severe constant headache, worse at night, situated in right parieto-occipital region; of vertigo and excessive vomiting, not dependent on taking food. He had cerebellar gait, usually, but not always, fell toward the right. Slight ataxia in the right arm was present when finger to nose test was attempted, but there was no weakness of cranial or spinal nerves. The reflexes were about normal, except that the patellar reflex was slightly exaggerated on the right side. No Babinski sign was obtained. He had diplopia at times. Nystagmus was seen in both eyes when he looked either to the right or to the left.

Examination.—Ocular examination was made by Dr. Fox Nov. 9, 1907. O. D. optic nerve decidedly red and vessels have pinched appearance as of optic neuritis. O. S., optic neuritis, slight hemorrhage just above optic nerve; slight paresis of left third nerve.

November 16.—Optic neuritis has greatly increased in both eyes. Veins exceedingly tortuous; apex of swelling, 7 D.

Treatment.—Operation by Dr. S. C. Burns Nov. 27, 1907. An opening was made in the occipital region, and the right lobe of the cerebellum was exposed, but nothing abnormal was found. The brain did not bulge unduly into the opening.

November 29.—Examination by Dr. L. Webster Fox. No change in the conditions found at examination on November 16. December 15: O. D., still pronounced swelling of nerve, with slight hemorrhages in striated form, showing through the outer halo of the circle, veins pronounced, arteries almost obliterated. Head of nerve intensely red. Probable change for the better. O. S., nerve still shows pronounced swelling. Hemorrhage of retina, down and out from optic nerve, extending to near equator as a broad band. Up and out on outer margin of optic nerve is a beautiful star-shaped hemorrhage, one-third the size of normal optic nerve.

December 9.—O. D., swelling of disc has diminished one-half. Arteries are thin. Veins still show some stasis. No hemorrhage. O. S., neuritis less pronounced. The center of the physiologic cup can now be seen. Surrounding optic nerve is a halo of white edematous retina, somewhat striated. The hemorrhage of the macular region has lessened. The veins downward and outward are markedly beaded. Both eyes show improvement.

December 15.—Examinations show about same conditions as on December 9. Distant vision, 20/50.

Jan. 1, 1908.—O. D., optic nerve on a level with surrounding retina. Slight haziness of margin showing some neuritis. Veins clearly defined and above normal in caliber, no blocking at entrance of nerve. Arteries still very thin, showing impeded circulation. Retina has an anemic appearance instead of being a bright red. Chorioidal arteries can be seen. Downward and outward in extreme periphery of fundus is a dark speck, impossible to say if a retina hemorrhage or not. O. S., same general appearance as O. D. Retinal hemorrhages so pronounced two weeks ago are entirely absorbed. General findings show marked improvement.

February 1.—O. D., optic nerve clear and distinct in outline. Blood vessels of normal caliber. No retinitis. O. S., same as O. D. Slight paresis of internal rectus muscles.

March 4.—After the operation the headache disappeared and the vomiting ceased. A swelling about the same of a half of a small orange, soft and fluctuating, appeared at the seat of the opening in the skull. The gait has become much more ataxic, the arms, especially the right arm, are also ataxic, so much so that he feeds himself with difficulty. Babinski's "adiadococinesia" is marked in the right arm. Speech is very thick and indistinct. Diplopia is marked and paresis of right third, sixth, seventh, fifth, ninth, tenth and eleventh nerves has developed.

Also some paresis of eighth nerve on the right side. Within the past few weeks the man has had some nausea in the morning and headache has returned, but not nearly so severe as before the operation. Eye sight is excellent for distance.

The question has been put to me several times whether palliative operation might be of benefit when the patient with signs of brain tumor has suddenly fallen into coma. Risien Russell, as already stated, is in favor of trephining when respiration has ceased but the heart continues to beat, even though the chances of saving life may be remote.

Very frequently, in my experience, sudden death has terminated the symptoms of brain tumor, especially when the tumor is in the posterior cranial fossa. The patient may be no worse than he has been for months, and suddenly he becomes cyanosed, unconscious, possibly has convulsive movements and respiration ceases, but the heart continues to beat. It has seemed extremely doubtful to me whether decompression would be advisable under such circumstances. Death is almost certain, and the physician who performs or recommends a palliative operation when the patient is at the point of death is likely to be regarded by the relatives as having hastened the fatal termination. Such operation, done at a more suitable time, I believe may materially lessen the danger of these sudden and usually fatal attacks.

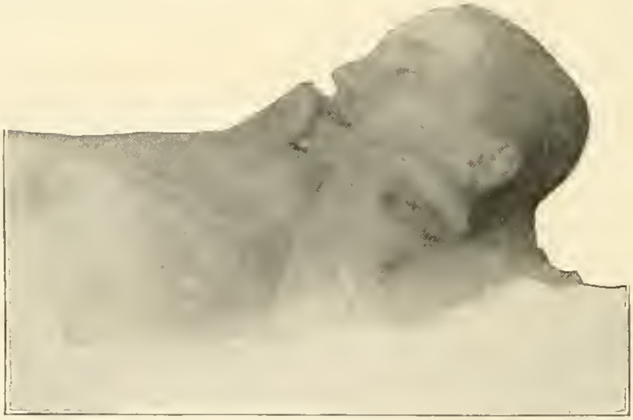

Fig. 1.—Showing the cerebellar position of upper limbs caused by a large tumor at the base of the brain displacing the cerebellum (Fig. 2).

A patient with brain tumor, who had also been under the care of several of my colleagues, recently died in my service at the Philadelphia General Hospital. A very large tumor was found in the posterior cranial fossa. The occipital lobes had been pushed apart and the cerebellum had been displaced almost to a right angle with the brain stem. Undoubtedly decompression in the posterior cranial fossa, by allowing space for the development of this tumor, would have removed much discomfort (Figs. 1 and 2). A case of sudden death, in which the question of palliative operation in the period of coma was considered, is the following:

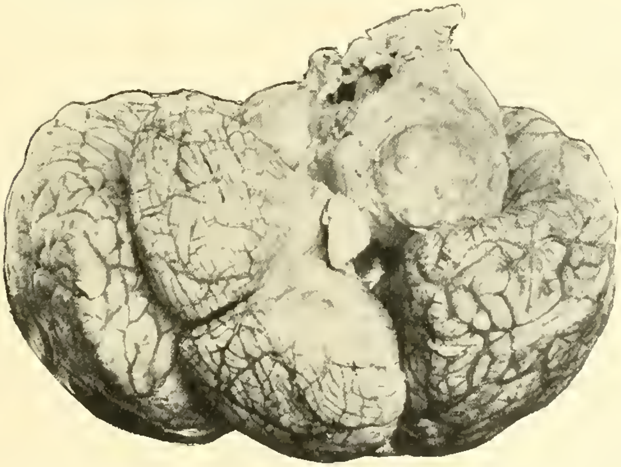

Fig. 2.—The tumor has twisted the cerebellum on its axis and pushed it between the occipital lobes.

Case 6.—*History.*—A man, aged 28 years, was referred to the University Hospital by Dr. J. W. McConnell Jan. 30, 1908. He had been complaining of headache during a year, of vertigo during two months, and of vomiting during one month. He was a brakeman and had been injured frequently. A year before admission he had had attacks of spasmodic movement in the left side of his body. In August, 1907, he had a sudden attack of twitching of the left side of the body, preceded by slight dizziness. He fell and probably was unconscious. He had had syphilis.

Examination.—On admission his gait was unsteady, and the staggering was increased by closing the eyes. The right pupil was larger than the left, and both irides reacted to light and in accommodation. The extraocular muscles were normal. The right nasolabial fold was not so deep as the left. Some ataxia was seen in the finger to nose test, especially on the right side. The right lower limb was stronger than the left. The right patellar reflex was exaggerated, the left slightly so. Ankle clonus was obtained on the right side, but not on the left side. A note made on February 1 is as follows: He has been complaining of severe headache, the face becomes flushed and the pupils dilated. He moans and cries and begs to be put out of his misery, but appears fairly comfortable most of the time.

I saw the patient February 4 about noon. He was suffering intensely from headache, threw himself about in the bed in an agony of pain, and caught hold of the resident when he came near him, begging to be killed and put out of his misery. He was quieted by a hypodermic injection. His suffering was so great that no examination could be made, and, knowing very little of his history, I did not feel justified in requesting an immediate palliative operation. About 3 o'clock in the same afternoon he was found on the floor unconscious, with very difficult breathing. He soon became cyanosed and respiration ceased. The heart action became slow and irregular. Artificial respiration was kept up until about 8:30 p. m., when death occurred. No necropsy was permitted.

I debated whether it would be advisable to have a palliative operation during the collapse, but it did not seem desirable. The chance of benefiting him thereby was very questionable. A similar case of brain tumor of one cerebral hemisphere under the care of Dr. Joseph Sailer was in the University Hospital about a year ago. Cessation of respiration occurred suddenly and artificial respiration was employed for several hours. A decompressive operation was performed by Dr. Edward Martin during the period of collapse without any benefit or any delay to the fatal termination.

Whether pain in itself can cause death or not I think is open to question, and it may be that the intense intracranial pressure with irritation may be the cause of the pain as well as of the sudden death, possibly by paralysis of the important centers of the medulla oblongata. Byrom Bramwell, however, believes that pain alone may kill. Where sudden headache is intense, as described in Case 6, and symptoms of increased intracranial pressure are present, it has seemed to me dangerous to delay decompression, and I regret that in this case it was not done, even though the clinical history was imperfectly known to me. This agonizing headache is sometimes too serious to permit any delay in giving relief by opening the skull.

About four weeks after the experience above related I was asked to see a case in consultation with Dr. Ernest LaPlace, in which symptoms of a focal cerebral lesion were said to have developed gradually within twenty-four hours. The patient, during my examination, had intense headache, and, fearing a fatal termination, I recommended immediate decompression. Trephining with slitting the dura was done and about two ounces of clear fluid escaped, with great temporary relief of the suffering within twelve hours. The opening probably was not sufficiently large.

Decompressive operation is not always beneficial, even when the patient is not in coma, and death may occur within twenty-four hours after a palliative operation, as illustrated by the following case:

Case 7.—*History.*—A. K., male, aged 25 years, was admitted to the University Hospital, March 4, 1907, complaining of blindness, intense headache and vomiting. His mentality was much impaired. He stated that during the winter of 1906-7 he began to have weakness and soreness about the articulation of his jaw. The soreness afterward spread upward in his head. About two months before admission, he became blind in the right eye, and two weeks later he became blind in the left eye. He vomited frequently. He was unable to walk without support, and could not stand with the feet together, but could stand if they were separated. The head was thrown far backward, and any attempt to move it forward caused much pain. The left pupil was considerably larger than the right, and neither iris reacted to light. Corneal and conjunctival sensation and the lachrymal reflex on the right side were lost, as well as the sensation of the mucous membrane on the right side of the nose. The sensations of touch and pain were completely lost in the entire distribution of the right fifth nerve, provided no pressure was made. The mouth deviated to the right, when opened. The patellar reflexes were lost. Babinski's sign was present on the left side.

Dr. de Schweinitz reported: Palpebral fissure about equal in width. Left eye slightly divergent. Left external rectus movement preserved. Movements of internal superior and inferior recti are markedly limited. No wheel movement obtained. Right eye: Loss of movement of external rectus and marked limitation of movements of superior and inferior recti, with almost lost internal rectus movement. There is extensive double optic neuritis with large retinal hemorrhages on the right side.

The case was believed to be one of tumor or syphilis of the base of the brain involving the right Gasserian ganglion.

Treatment.—A decompression operation was done by Dr. Frazier on each side of the head on April 11, 1907, just above the ear. The brain bulged much on the right side. The patient died the following day. An endothelioma was found at the necropsy, implicating the right Gasserian ganglion. Decompression in this case did not prolong life, and it may be, hastened the fatal termination.

In the following cases decompression was of benefit:

Case 8.—*History.*—Ida K., a woman, aged about 21, consulted me Nov. 3, 1906. She complained of failure of vision during two months. She had aborted four months previously, and headache began about eight days after abortion. It was sometimes frontal, sometimes occipital, and frequent. She had not had nausea, vomiting or convulsions. Menses returned after the abortion and had been regular.

Examination.—The lower part of the right side of the face was weak in showing the teeth; the upper part of the face was not affected. She could not draw up the right side of the mouth separately. The tongue deviated slightly to the right. The grasp of the right hand was weaker than that of the left. The right lower limb seemed a little weaker than the left in resisting passive movements. The patellar reflex seemed to be exaggerated on the right side. The patient later came under the care of Dr. Mills.

An examination of the eyes by Dr. de Schweinitz, Nov. 27, 1906, revealed double choked disc, no external muscle palsy, and very contracted fields.

Treatment.—Dr. Frazier performed a palliative operation Nov. 28, 1906. An osteoplastic flap was made extending one inch behind and three inches in front of the Rolandic fissure. When the dura was exposed, it was found exceedingly tense, and when it was cut, the brain bulged greatly. The veins of the pia were markedly distended. After making a transverse slit in the dura under the middle of the flap, the dura was closed with interrupted silk sutures. A portion of the temporal bone below the flap was then rongeured away for decompression. The skin was closed with interrupted sutures. Hernia cerebri was observed December 6.

The report of Dr. de Schweinitz, December 9, is as follows: Right Eye—Optic neuritis apparently subsiding, + 5 D. (compared with + 7 D. before operation). Both sets of vessels very small. In both maculæ there is some chorioidal disturbance. Left Eye—Optic neuritis + 5 D. Vision (uncorrected), each eye 4/9.

On December 11, the note was made that the patient did not have headache as before the operation and did not vomit. The patient was dismissed from the University Hospital and was admitted to the Philadelphia General Hospital, service of Dr. Mills, Oct. 17, 1907. An examination was made by Dr. Shumway October 22. Pupils respond very sluggishly, are well dilated. Both nerves show advanced optic atrophy following choked disc. The nerve heads are still swollen to a level of 6 diopters (2 mm.) above the retinal level. The macular regions show no changes. The hernia had increased considerably in size.

Death occurred Dec. 7, 1907. A large glioma was found in the left temporal lobe, growing over the left cerebral peduncle (Fig. 3).

The improvement in the condition seems to have been of a duration of months, but as she was not constantly under observation the exact time is uncertain.

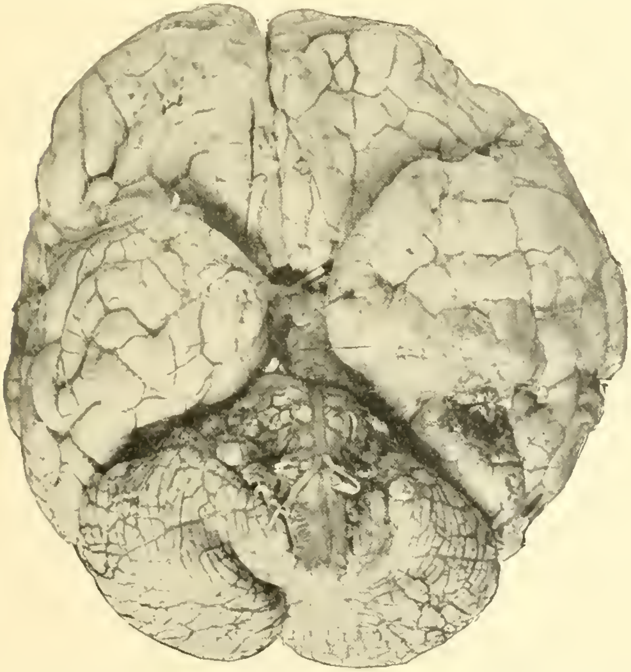

Fig. 3.—A large glioma occupied the greater part of the left temporal lobe and grew as a wedge-shaped process over the left cerebral peduncle (indicated by the line). The left temporal lobe was much enlarged.

Case 9.—*History.*—A. B., 42 years of age, consulted me June 28, 1906. He had an enlargement of the skull on the left side above and in front of the ear. He complained of weakness on the right side of the body, slight ataxia, some motor aphasia, left-sided headache, some difficulty in reading, and failure of memory.

Examination.—Dr. de Schweinitz found no evidence of optic neuritis, but there was slight over-fulness of the retinal vessels.

Treatment.—Decompression without opening the dura was performed, July 7, 1906. Almost complete disappearance of symptoms followed for about two months, and possibly the improvement would have lasted longer if the dura had been opened. A later operation revealed an endothelioma, which was removed. A full report of this case has been published.[15]

The following case was in the service of Dr. Morris J. Lewis. The operation was not palliative, but was removal of a tumor. It is included because it shows remarkable improvement in the ocular conditions after the operation.

Case 10.—*History.*—K. H., aged 33, a patient at the Orthopedic Hospital. July 10, 1907, complained of convulsions for past three years. Screams, falls to floor unconscious, bites the tongue. Attack begins in the left arm. She has had about forty attacks in the past ten years and they are becoming more frequent and severe. The eyes were found normal, except for the error of refraction, + 1.5 D. at this visit.

Examination.—Sept. 30, 1907.—Seen by Dr. S. D. Risley at his office; no fundus changes.

October 9.—Seen by Dr. Langdon, and beginning optic neuritis is discovered; discs swollen, 2 D. above fundus level; margins blurred; veins full; no hemorrhages. Fields and vision normal. Brain tumor suspected.

October 19.—Admitted to house. Attacks continuing; vision, fields, muscles and pupils normal; discs swollen; O. D., 3 D. above fundus; O. S., 2.5 D. above fundus. Veins more full and tortuous; numerous transudations of serum, from veins; no hemorrhages.

October 25.—No increase in swelling of discs, numerous hemorrhages in O. D.; none in O. S.

November 4.—Discs swollen, 4.5 D. Several large hemorrhages in O. D. Transudations on O. S. Vision full.

November 15.—Dr. Taylor operated, removing a large tumor (glioma) from upper posterior border of the fissure of Rolando. Patient made an uneventful recovery, with a left hemiplegia.

November 16.—Eyes deviated to right.

November 17.—O. D. disc, 2.5 D. above fundus, and O. S., 1.5 D.

November 22.—O. D., 1.5 D.; O. S., 0.5 D.

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[15] Spiller, W. G.: The Journal A. M. A., Dec. 21, 1907, p. 2059.

November 24.—Seen by Dr. de Schweinitz and last measurement verified.

December 3.—O. D. disc 1.5 D. above fundus. O. S., no swelling, margins somewhat blurred. No hemorrhages; vision full.

I have attempted to learn the present condition of the patients still alive reported in the previous paper by Dr. Frazier and myself, on whom decompression was performed. This and the results of experimental work will be reported later.

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BRAIN TUMOR*

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During the past year I have had the opportunity to observe a number of cases of brain tumor, certain features of which seem to me worthy of discussion. Especially noteworthy are the importance of gradually developing hemiplegia, the slow appearance of papilledema in certain cases of glioma, and later its remarkably rapid development, the resemblance of tumor of the pons to tumor of the Gasserian ganglion, and the signs of tumor of the occipital lobe, especially the dissociation of the color-sense in tumor of the left occipital lobe.

DIAGNOSIS OF BRAIN TUMOR

The credit for pointing out that progressive hemiplegia without other symptoms is diagnostic of brain tumor seems to belong to R. T. Williamson. In his paper[1] he states that it is well known that when hemiplegia is produced by a tumor in the motor cortex or white matter of one cerebral hemisphere the onset of the paralysis is usually very gradual. Day by day, and week by week, the loss of power becomes more and more marked. It may be weeks, or a few months, before the paralysis is complete. It is not so well known, he states, that progressive hemiplegia of the nature just described is an almost certain sign of brain tumor, even in the absence of optic neuritis, headache, and vomiting (provided there are no indications of cerebral abscess). Williamson reports three cases of brain tumor in which the hemiplegia developed very grad-

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* Read in the Section on Nervous and Mental Diseases of the American Medical Association, at the Sixtieth Annual Session, held at Atlantic City, June, 1909.

[1] Williamson, R. T.: Practitioner, July-December, 1904, p. 321.

ually, and in all three papilledema was absent. In the first case headache and vomiting were present; in the second case these two symptoms were absent for a long period; in the third case they were absent altogether. He refers to a similar case observed by him ten years previously. He expresses his views very clearly: "Provided there are no indications of cerebral abscess, progressive hemiplegia, or hemiplegia of gradual onset, in which weeks or a few months elapse before the paralysis is complete, may be regarded as strong evidence of cerebral tumor, even when optic neuritis is absent, or when optic neuritis, headache, and vomiting are all absent."

I can not refrain from stating that Williamson expresses his opinion somewhat too forcibly. A gradually developing hemiplegia I have seen as a symptom of polioencephalitis. A child recently under my care presented symptoms suggestive of brain tumor. An operation was performed at my request by Dr. Frazier and a portion of the cortex was removed, as at the operation it resembled tumor tissue. It proved to be the seat of polioencephalitis. Six weeks before the child came under my observation the right lower limb began to get weak, and one week later the right upper limb began to grow weak. The hemiparesis became gradually worse. The case I shall probably report in detail, and merely wish to call attention here to the fact that polioencephalitis as well as tumor may cause a gradually developing hemiplegia. The latter lesion is, of course, of much more frequent occurrence than the former.

Souques[2] also has recently written on gradually developing hemiplegia as a sign of tumor.

Cortical meningitis may cause a gradually developing hemiplegia, and the meningitis may be purulent or of other type. I have recently seen a case with Dr. W. G. Shields, Jr., and Dr. J. A. Hearst in which paralysis of the left lower limb, almost confined to the part below the knee, developed one day, and paralysis of the left upper limb and left side of the face did not appear until two days later. An operation by Dr. Edward Martin revealed a purulent meningitis, apparently confined to the region of the center for the lower limb. The paralysis in such a case, however, is likely to be of more rapid development than when caused by tumor.

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[2] Souques: Rev. neurol., 1908, p. 1250.

The gradual development of hemiplegia was present in several of the cases mentioned in this paper. It was especially striking in Case 1, and, indeed, because of it I made the diagnosis of a probable tumor of the brain at my first examination of the patient. A hemiplegia had developed very gradually during a period of about six weeks, and there was at no time a sudden increase in the paralysis. Even within three days of death no changes in the eyegrounds were observed in an examination by Dr. de Schweinitz. A large tumor was found in the optic thalamus and internal capsule, and had undergone cystic degeneration. It had evidently caused no increase of intracranial pressure. Cases such as this and Cases 2 and 3 indicate that the production of papilledema by toxic substances alone in brain tumor is questionable. In all these cases the tumor was large, and yet papilledema did not occur, in two cases and developed only late in the other, probably because there was no great increase of intracranial pressure.

In Case 2 a gradually developing hemiplegia was observed, and yet the tumor did not extend to the internal capsule. Vascular changes in the internal capsule at a distance from the tumor caused the hemiplegia, as determined by the examination of certain sections given by me to Dr. Byrnes for this purpose. The importance of this finding is in the demonstration that a gradually developing hemiplegia in brain tumor does not imply necessarily that the tumor has extended to the internal capsule. The examination of the brain with the clinical notes in mind was a surprise to me, as to the naked eye no implication of the internal capsule could be detected, and the tumor evidently did not extend so far forward from the occipital lobe. Dr. Byrnes[3] has referred to these findings in another paper.

A gradually developing hemiplegia was observed also in Case 4, but the patient was not seen by me until the symptoms were far advanced, and it was uncertain how much reliance could be placed on the statements.

In a case seen by me in consultation with Dr. Babcock a diagnosis of brachial plexus palsy had been made at first, because the palsy seemed at that time to be confined to the upper limb. At the time I saw the patient the lower limb had also become affected, and other symptoms made the diagnosis of tumor a probable one. A cortical tumor was found by Dr. Babcock at operation.

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[3] Byrnes: Jour. Nerv. and Ment. Dis., March, 1909.

I have in two instances known thrombosis of the middle cerebral artery to cause a gradual increase in the symptoms as seen in tumor, but when thrombosis is the cause the development of the symptoms is likely to be somewhat different. A partial palsy, or other symptoms indicative of brain lesion, may develop within a short time, and then the symptom-complex may be arrested for some time, possibly several weeks, until the thrombus extends to one or more branches of the vessel previously affected, when additional palsy appears, but usually more rapidly than is seen when a tumor is the cause of the weakness. Occasionally a tumor may produce a very rapid palsy, as when a hemorrhage implicates the growth or when a glioma has destroyed many of the axis cylinders within the tumor but has left enough for the discharge of function, and suddenly, by edema or other cause, the impaired fibers become insufficient.

A peculiar symptom, present in Case 3, I have observed in two other cases of brain tumor, viz., intense hunger. Mrs. Y was hungry all the time and had a "most vigorous appetite," in the words of her friends. In another case a note was made that the patient had a voracious appetite. The intense hunger is not confined to tumor cases. The character of the tumor seems to have no bearing in the production of this symptom, nor does its location seem to have much importance.

Mrs. Y. (Case 3) also complained of pain and throbbing extending down the neck to the shoulders. Pain from tumor of the brain may be felt in the neck. This neck pain was very pronounced in a case reported by me some years ago, and in that case the tumor was at the foramen magnum and extended downward on the cord the distance of several cervical segments. Why pain should be felt in the neck from a tumor of the brain is not clearly understood, any more than is the related symptom of stiffness of the neck observed sometimes in cases of brain tumor, as in Cases 1, 2 and 4, in which rigidity of the neck muscles was caused by a tumor of the optic thalamus, by a tumor of the occipital lobe, and by a tumor of the region posterior to the optic thalamus. So far as I am able to determine, pain radiating down the neck and stiffness of the neck are not of localizing value.

The slow development of papilledema in glioma is worthy of comment. It may be absent throughout the disease, even when the tumor is large, as in Case 2, in which almost the entire occipital lobe of one side was occupied by a tumor, or in Case 3, in which it developed late, and after the tumor had attained a very large size. The latter case demonstrated another interesting phenomenon, viz., the occasionally extraordinarily rapid development of papilledema in brain tumor, even though it may be late in appearing. This patient was found on March 21 to have normal eyegrounds; on April 6 Dr. Thorington noticed for the first time that the nasal side of each disc had begun to be elevated and to be indistinct in outline, and that the retinal veins were enlarging. On April 13 he found the swelling of the discs to amount to 7 D., and on April 18 to 10 D. The case shows how important constant observation of the eyegrounds may be when the symptoms of brain tumor are present, and illustrates the danger of delaying decompression when the signs of increased intracranial pressure appear. It is often impossible to make the patient realize that the sight may be put in jeopardy within a few days, and he may neglect to visit the ophthalmologist, notwithstanding that every effort may have been made to convince him of the danger of this neglect.

Paton[4] has studied the records of 252 cases of brain tumor occurring at the National Hospital between the years 1899 and 1905. He finds that tumors of the deeper portions of the cerebral hemispheres and of the pons are more likely to occur without papilledema. In the subcortical cases the development of papilledema almost invariably corresponded with invasion of the cortex or with marked compression or invasion of the lateral ventricle. The development of papilledema in pontile tumors almost invariably implied the invasion of the cerebellum.

In the discussion of this paper Gunn[4] states that pontile tumors rarely cause papilledema because death occurs early.

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[4] 4. Paton and Gunn: Tr. Ophth. Soc. U. Kingdom, 1907-1908, xxviii, p. 112.

The statements quoted from the remarks of Paton conform with the results of my own observations in great measure, but the explanation offered by Gunn for the infrequency of papilledema in cases of pontile tumor is inadequate. I have repeatedly seen tumors causing enormous enlargement of the pons before death occurred, and in Case 3 the tumor of the pons doubtless was enormous before papilledema occurred. The cerebellum was not implicated, and, notwithstanding Paton's statement, papilledema became intense. Likewise, in Case 2, the tumor occupied almost the whole of the occipital lobe, but caused no papilledema, even though it extended to the cortex. Rules are useful, but almost all have exceptions, and what is true of one series of cases is not true of all.

Some years ago Schmidt[5] advanced the view that an increase of the symptoms by lying on a certain side of the body was indicative of the side of the tumor. The sign has seemed to me exceedingly unreliable, and I have been observant of it since the first appearance of Schmidt's paper, but have never been able to obtain any satisfaction from it, and have come to entirely disregard it from a diagnostic point of view. Mrs. Y (Case 3), for example, was obliged to lie on the right side, and when she turned on the left side she complained of feeling sick and as if "everything were going from her." Turning the head to the left while lying down produced nausea. In this case the tumor implicated the entire pons.

In Case 5 the diagnosis had to be made between tumor beginning in the Gasserian ganglion and tumor of the pons. The patient had headache, vomiting, vertigo when headache was present, and diplopia. In my examination I found that he had considerable ataxia, slight nystagmus in looking far to either side, slight, weakness of the left facial distribution, some impairment of hearing in the left ear, and marked paralysis of the left trigeminal nerve (atrophy of the temporal and masseter muscles with diminution but not complete loss of sensation in the distribution of this nerve) and paresis of the left side of the soft palate.

One might readily diagnose a tumor of the pons with these symptoms, but the examination also showed that the voluntary power of the upper and lower limbs on each side, as well as the tendon reflexes of these limbs, was normal. The ocular examination revealed beginning papilledema and paresis of the left external rectus. The deafness of the left ear was attributed to catarrh, as no signs of nerve deafness were detected.

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[5] Schmidt: Wien. klin. Wchnschr., 1898, No. 51.

Probably the most striking sign was the pronounced implication of the left trigeminal nerve, so intense as to cause a distinct flattening of the face over the position of the muscles supplied by this nerve; and this would seem to indicate a long-standing implication of the fifth nerve. A tumor of the Gasserian ganglion would explain this sign, as well as the escape of the motor tracts for the lower limbs. The left sixth nerve might easily be caught in its passage through the cranium. Pressure of the tumor on the pons—i. e., on the middle cerebellar peduncle—might explain the nystagmus on lateral movement and the ataxia. A tumor in this position might cause some weakness of the left facial nerve and beginning deafness. I am inclined to think that central deafness at first may be indistinguishable from deafness due to middle-ear trouble, and that we should be careful how we diagnose when deafness is in an early stage. The weakness of the left soft palate was difficult to explain by the supposition of a tumor beginning in the Gasserian ganglion and extending downward sufficiently to implicate the glossopharyngeal nerve, but no less difficult appeared the diagnosis of tumor within the substance of the pons sufficiently large to implicate the fifth, sixth, seventh, eighth, and possibly the ninth and tenth nerves, and yet cause no weakness of the limbs or change in the tendon reflexes. The diagnosis was extremely difficult and could not be made with certainty, and, in the hope that the tumor might be found in the region of the Gasserian ganglion, although pain in the face had not been severe, I requested Dr. Frazier to operate, but the tumor was not detected at the operation. It is indeed remarkable that a tumor so large as that found in this case could occupy a large portion of the pons and yet produce no weakness of the limbs or change of reflexes that could be detected in careful examination directed toward the finding of either of these signs. The explanation is that the growth was a glioma, and, as so often occurs in tumors of this variety, many nerve fibers passed through the tumor without losing their axis cylinders, even though they may have lost their medullary sheaths.

Pain in the distribution of the fifth nerve would be more likely to occur in tumor of the Gasserian ganglion.

The case shows clearly that the diagnosis between a tumor upon the pons and one within the pons, if the latter be a glioma, my be extremely difficult or even impossible. A case presenting the same difficulty I studied with Dr. W. A. Bliss in St. Louis somewhat over a year ago.

In my chapter on tumors of the brain, in the book edited by Dr. Posey and myself,[6] I give the symptoms that have been observed in tumors of the occipital lobe. Visual hallucinations are mentioned, and several references are given to cases in which these phenomena occurred. The hallucinations seem to occur in the blind fields, and this statement is also made by Oppenheim.[7] The appearance of homonymous lateral hemianopsia, followed by gradually developing hemiplegia and loss of sensation, all the symptoms being on the same side, is suggestive of tumor, especially if the hemianopsia be accompanied by visual hallucinations. The latter may be absent throughout the course of the disease; thus Claude and Rose[8] have recently reported two cases of tumor of the occipital lobe in which visual hallucinations were not observed. They assume that such hallucinations are caused by irritation of the calcarine cortex, and were absent in their cases because the tumors were in the white matter of the occipital lobe. This seems to be assuming too much, as we have sufficient evidence to show that a lesion need not be confined to, or even be located within, the calcarine cortex to produce visual hallucinations, and this statement is justified by Case 6. I have emphasized this fact also in my chapter referred to above. Claude and Rose refer to the fact that the hemianopsia in tumor of the occipital lobe is seldom complete from the beginning, but, after being partial for a time, may rapidly become complete, so as to suggest a thrombotic lesion. In one of their cases an examination made Jan. 9, 1907, revealed a left homonymous lateral quadrantanopsia. Another examination, made Feb. 24, 1907, showed that the hemianopsia had become complete.

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[6] Posey and Spiller: The Eye and Nervous System, 387.

[7] Oppenheim: Die Geschwülste des Gehirns, ed. 2, p. 120.

[8] Claude and Rose: First Internat. Cong. of Psychiat., Neurol., etc., Amsterdam, Sept. 2-7, 1907.

All writers who refer to visual hallucinations in cases of brain tumor, so far as I have been able to study the literature on the subject, speak of the hallucinations as occurring on the side opposite to the tumor. This generally accepted opinion led me to a diagnosis of the wrong occipital lobe in a case seen with Dr. William H. Teller (Case 6). The patient stated positively that he had frequent attacks, in which wriggling bright lights, beginning always in the right field, gradually passed to the center of the field, and disappeared in the left field. These phenomena were accompanied by dimness of vision, beginning also in the right field and disappearing in the left field. Since optic neuritis had occurred he saw black objects looking like snakes, rosettes, and forms of leaves in front of the eyes; occasionally he saw the heads of persons. Believing from these phenomena that the tumor was probably on the side opposite the fields in which the hallucinations first appeared, and therefore in the left occipital lobe, I suggested to Dr. Teller that it would be well to expose the left occipital lobe. The tumor, however, was in the right occipital lobe. It would seem from this case that visual hallucinations may occur in the field on the same side as the tumor, or at least begin in this field. The patient was intelligent, his mind was active, and his statements regarding his visual phenomena were reliable.

The only important signs of tumor in this case were papilledema of high grade and visual hallucinations. Hemianopsia was not present, and the absence of this sign is explained by the examination after death, as the tumor does not appear to have implicated the visual fibers. It seems almost impossible to have made any other localization than that made in this case, unless we can judge from one case that visual subjective phenomena may occur in the field on the same side as the tumor.

Another case seen with Dr. J. T. Rugh more recently lends support to this view. A woman with signs of brain tumor saw flowers and "red, green and bright colors," and dogs, to her right, and on one occasion she saw bird-cages and an open trunk, but it is not certain where she saw these; the flowers and dogs she was sure she saw to the right. Spontaneous pain in the right parieto-occipital region and tenderness to percussion here, normal right patellar reflex and exaggerated left patellar reflex, and some impairment of ability to recognize objects with the left hand, indicated that the lesion was on the right side. The tumor was not found at operation, but the pressure of the right cerebral hemisphere through the opening was enormous.

Lewandowsky[9] refers to the amnesic color-blindness of Wildbrand. This seems to be a sensory aphasia confined to the names of colors. Patients presenting this sign are unable to name colors shown them and to pick out colors named to them. Lewandowsky is uncertain whether this symptom occurs in a pure form, and is inclined to think that the symptom-complex belongs to his dissociation of the color-sense. His patient had sensory aphasia, was word blind and word deaf, but after these symptoms had almost disappeared the peculiar color-symptoms were detected. He had homonymous right lateral hemianopsia. Like my patient referred to below, the man was not mind-blind. My patient had no difficulty in recognizing and avoiding objects. Lewandowsky's patient was unable to name the colors of objects shown to him or to find colors named to him. He could not name the color of an orange or a leaf (my patient could not name the color of blood or of snow). He also could not find the proper colors of objects from samples shown him, as my patient also could not. The disturbance evidently did not depend merely on a speech defect. His patient also did not observe anything peculiar in pictures colored unlike objects occurring in Nature. *He was able to match colors.*

Lewandowsky believed that a lesion had destroyed the color-center of the left hemisphere, but that the color-center of the right hemisphere was preserved. The association of the color-sense not only of the left center but also of the right center was through the left color center, and, this being destroyed, association of color with form was impossible. Whether the left occipital lobe is the more important in every person is not determined, but in all reported cases of cerebral disturbance of the color sense the left hemisphere has been affected.

It would seem that in this dissociation of the color-sense described by Lewandowsky[9] we have a new and important sign of lesion of the left occipital lobe. It is possible that it might occur without other sign of lesion of the occipital lobe, but it is probable that we shall always find hemiamblyopia, as in my case, or even hemianopsia.

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[9] Lewandowsky: First Internat. Cong. Psychiat., Neurol., etc., Amsterdam, 1907; Monatsschr. f. Psychiat. u. Neurol., 1908, xxiii, 488.

A patient in the service of Dr. John H. Musser, whom I had the opportunity to examine, presented this symptom of color disturbance, which promises to be of value in diagnosing lesions of the left occipital lobe. It is not my intention to enter into a full description of the case in this place. He had right lateral homonymous hemiamblyopia, intense word-blindness, slight word-deafness, and some amnesic aphasia, i. e., he could not always find the word he desired in speaking. As there was a possibility of tumor, although the lesion was believed to be thrombosis, I recommended exposure of the left occipital lobe with the angular gyrus as a center, and softening of the occipital lobe was found by Dr. Frazier and the diagnosis confirmed by the examination of a small piece of excised tissue. An examination made by me before the operation gave the following results:

The patient could name most objects shown to him, such as a watch, knife, key, handkerchief, but sometimes made mistakes. When shown the picture of a dog colored deep blue or the picture of a donkey colored deep green he found nothing peculiar in these colors, and said when asked that he had often seen a dog or a donkey of the colors mentioned. He was entirely unable to give the name of any color shown him, and when the correct name of the color was mentioned among others he did not recognize it. He could match colors without the slightest hesitation. He was entirely unable to find a color mentioned to him when a variety of colors was placed before him. He was unable to name the color of any object shown to him, as that of a leaf. He answered, "I know what all that is, but I can not remember." When asked what a leaf was he said that he did not know.

A hasty examination would have led to the conclusion that the patient was color-blind, as he did not remember the color of any object, did not name the colors shown him, did not see any inpropriety in the coloring of a dog blue or a donkey green, and yet the preservation of the color-sense was shown by his ability to match colors without the slightest hesitation. He had merely lost the ability to associate color with form; he recognized the form of most familiar objects and matched colors, but was entirely unable to put form and color together. He had what Lewandowsky has described as *Abspaltung des Farbensinnes*, which might be translated as dissociation of the color-sense.

It is usually difficult or impossible to form an opinion of the character of the tumor from the symptoms. As regards the possibility of this, Oppenheim[10] says:

> The symptoms are not essentially modified by the character of the growth, especially when the syphilitic process is left out of consideration. The same symptom-complex may be produced by glioma, sarcoma, solitary tubercle, and other tumors. The glioma sometimes is characterized by apoplectiform attacks, which may occur repeatedly and be associated with a rise of temperature. The vascularization may cause much variation in the intensity of the symptoms. The glioma has a slow growth, excepting the acute attacks caused by hemorrhage and swelling. Remissions are not uncommon.

Ludwig Bruns[11] speaks of the uncertainty in diagnosing the character of the tumor from the symptoms, but he thinks that very acute commencement of symptoms, possibly after slight trauma, in a person previously apparently entirely healthy, is suggestive of glioma. Rapid variation in the intensity of the symptoms and sudden increase in the symptoms are also common in glioma, but occur in other forms of tumor. The diagnosis between glioma and sarcoma especially is difficult.

There is still another way by which we may draw conclusions regarding the presence of glioma: The general symptoms of brain tumor, associated with the signs of a widespread lesion as of the greater part of the pons, with the escape of important fibers situated within the lesion, as those of the pyramidal tract, should be indicative of glioma. In no other form of tumor is the escape of fibers within the tumor so likely to occur. In two of the cases reported in this paper the justification for this view is clearly presented (Cases 3 and 5).

REPORTS OF CASES

CASE 1.—*Patient.*—D. P. (Lab. No. 467) was referred to me Jan. 11, 1909, by Dr. S. W. Gadd. He had been well until six weeks previously. He denied venereal disease, and his wife had not had any miscarriages. A left hemiplegia had developed gradually, there had not been at any time a sudden loss of function, for example he was weaker at the time I saw him than he had been two weeks previously. He had not taken alcohol for twelve months, previous to this time he drank occasionally. He had not had headache, and gave no history of trauma. During the five days previous to his visit to me he had vomited almost every day. He had not had convulsions.

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[10] Oppenheim: Die Geschwülste des Gehirns, ed. 2, p. 221.

[11] Bruns, Ludwig: Die Geschwülste des Nervensystems, ed. 2, p. 260.

First General Examination.—The notes concerning his condition at my first examination are as follows: "Mentality is good, and he talks fluently. The sight has failed during the past two years. He has slight weakness in the lower left side of face, not in the upper part of face. The tongue is protruded in the median line. The left upper and lower limbs are weak, but he still has considerable power in these limbs. The biceps and triceps tendon reflexes are a little exaggerated on the left side. The patellar reflexes are prompt, and the left is distinctly exaggerated. Ankle clonus is present on the left side, but not on the right side. There is no hemianopsia. Sensations of touch and pain are less acute on the whole left side; in face, upper and lower limbs. He has marked tremor of the right hand. He drags the left foot when walking. He has no hemianopsia."

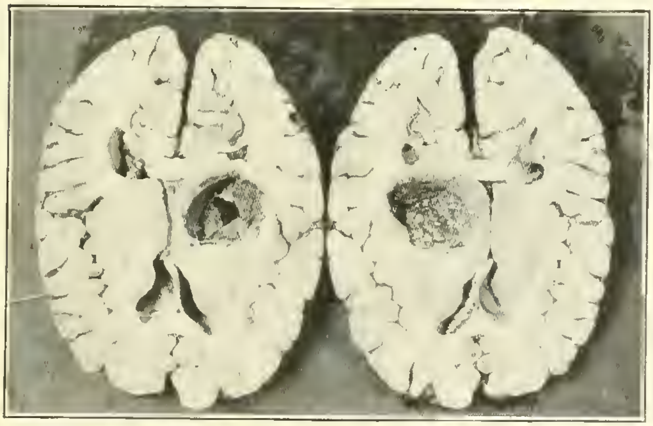
Fig. 1.—Case 1. Adenocarcinoma of the right optic thalamus and adjoining region.

Second General Examination.—The change that occurred in this patient's condition between January 11 and February 6 was very great. An examination made by me on the latter date gave the following results: "The man is in stupor; he mutters constantly, and makes irrelevant remarks. Hiccough is pronounced. He lies with his head turned to the right and the neck is stiff. The palsy in the lower part of the left side of the face is distinct. The stupor prevents any careful testing of the power of the upper part of the face. It is impossible to get him to protrude the tongue beyond the lips, or to make him move the eyeballs on command. The reaction to light is present in each eye, but feeble. He moves the right upper limb freely. The left upper limb is completely paralyzed, is flaccid, and falls lifeless when elevated. The right upper limb is somewhat spastic. The biceps and triceps tendon reflexes are about normal on each side, although they may be a little prompter on the left side. He seems to feel pin-prick in the left hand. The left lower limb is completely paralyzed, and falls lifeless when elevated. The patellar reflex is about normal on each side. Ankle clonus is not obtained on either side, and the Achilles reflex is weak on the left side. The Babinski reflex is uncertain on each side."

Ocular Examination.—The notes dictated by Dr. de Schweinitz regarding the ocular condition are as follows: "Pupils dilate thoroughly with homatropin. Right disc shows no swelling or edema, outer half has atrophic pallor. No definite signs of previous neuritis. Central vessels are small. The right disc is similar to the left, but of better color. In both maculæ patches of retinochorioiditis."

The man's condition became gradually worse, and he died February 10, at 6 a. m., his temperature, which had been irregular while he was in the hospital, rising to 106 just before death. A tumor was found in the region of the right optic thalamus.

Autopsy.—When the ventricles of the brain were opened after death no excess of fluid was found within them. The right optic thalamus fluctuated as though it contained fluid, and when a horizontal section was made through the cerebral hemisphere a cyst was found in the region of the upper part of the right optic thalamus and inner capsule. Twelve c. c. of cloudy yellow fluid were collected from this cyst. The tumor caused considerable enlargement of the right side of the brain, as shown by a horizontal section through both hemispheres at the level of the upper part of the optic thalamus. At this level it measured 4 cm. from before backwards and transversely. The cyst occupied the entire center of the tumor, leaving a narrow wall of tumor tissue. The growth was well defined from the surrounding tissue to the naked eye, and did not have an infiltrative appearance, nor did it extend into the lateral ventricle, but was separated from it by about 2 cm. of what appeared to be brain tissue.

Histologic Examination.—Under the microscope the tumor had a papillomatous appearance. It was sharply defined from the brain tissue, but extended into it by numerous nests of cells. It was very vascular and many of the vessels were surrounded by a row of cylindrical or somewhat cuboidal cells, each having a nucleus at the end nearer the vessel, the cells resembling the lining cells of the ventricle. The vessels were limited by a row of endothelial cells and separated usually by a space from the surrounding row of columnar cells. The tumor contained much detritus and some fatty granular cells.

It had the appearance of adenocarcinoma, and seemed primary. A squamous-cell carcinoma was found in the esophagus, but it is questionable whether the tumor of the brain could be considered as a metastatic growth from the tumor of the esophagus, especially as it was of a different type of carcinoma, and was solitary in the brain. The necropsy was performed by Dr. Karsner.

A very similar tumor of the brain has been described recently by Kölpin.[12] It was papillomatous and was regarded as an adenocarcinoma. It resembled glandular tissue where the tumor processes were cut transversely. It was sharply defined from the surrounding nervous tissue, but caused no distortion of the tissue about it, so that the growth of the tumor was proportional to the destruction of the nervous tissue. Masses of detritus were found in the center of the tumor, whereas the center of the tumor in my case had become cystic and the tumor had caused enlargement of the hemisphere.

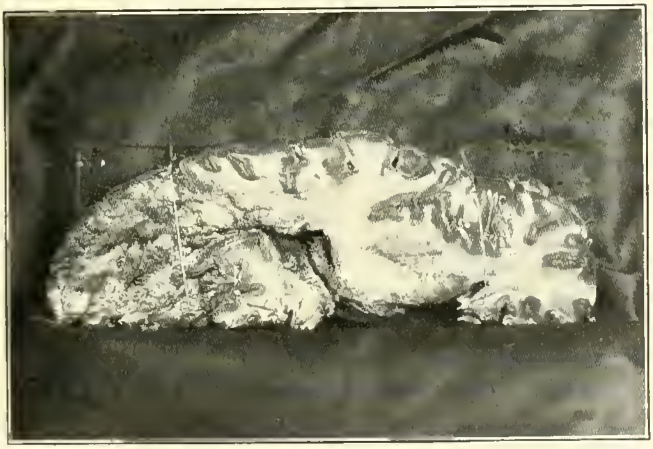

Fig. 2.—Case 2. Glioma of the left occipital lobe.

Primary epithelial growths of the brain, Kölpin states, are rare findings. They arise in the ependyma of the ventricles or the lining of the chorioid plexus and have a papillomatous formation. Kölpin refers to a number of reported cases. He believed that the largest of his tumors was the primary growth and that it originated in the ventricular wall, although such a connection could not be positively detected, any more than it could be in my case.

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[12] Kölpin: Arch. f. Psychiat., xlv, No. 2, p. 595.

Case 2.—*Patient.*—M. Sher. (Lab. No. 448) was admitted to the service of Dr. Edward Martin, at the University Hospital, April 23, 1908. I report the case with his permission. The patient's age was 53. The history of his illness was obtained from his wife. He was said to have been kicked by a horse in the head during the summer of 1907, but was not disabled at the time. He had not been sick during the past twenty-eight years, and he had not been a drinking man. He had been complaining occasionally for some months of headache, and his wife had noticed drowsiness, which gradually increased until he stopped work nine weeks previously. At that time he complained of severe headache in the left parietal region and seemed confused. The headache was persistent. During the four weeks previous to admission he had been in bed and was stuporous most of the time, though occasionally rational for short periods. The right arm was said to have become paralyzed four days prior to admission, and the right leg about a week before admission. The paralysis of the lower limb had developed gradually. Further investigation made the duration of the stuporous condition as given doubtful, and it seemed probable that it may have begun only a few days before he entered the hospital. When he entered the hospital he was stuporous all the time.

Ocular Examination.—Dr. Shumway made the following report: " Pupils are equal and respond to light. There is much mucopurulent discharge, obscuring the cornea. He has bilateral incipient cataracts. Retinal veins are full, but not tortuous. There is no blurring of tne nerve edges, no neuritis. The eyegrounds show no gross lesions."

General Examination.—An examination by me April 25 gave the following results: "Once when he was told to show his teeth he did so, and the nasolabial fold appeared to be as deep on one side as on the other. When the face is pricked with a pin on either side the corner of the mouth is drawn up, and there is no distinct evidence of facial palsy. He has a tendency to repeat in a whisper what is said to him. Pin-prick evidently causes pain in the face. Tactile sensation can not be tested anywhere because of the mental state. He partially understands what is said to him. The neck muscles are distinctly rigid. The right upper limb appears to be completely paralyzed; pricking with a pin in this limb causes some movement of the left upper limb, but none of the right upper limb, and the pricking is not correctly localized. The right upper limb seems to have impaired sensation. The biceps and triceps reflexes are about normal in the left upper limb, but somewhat exaggerated in the right upper limb. Testing with a feedingcup seems to indicate that the man has right hemianopsia. The patellar reflex is much diminished on the left side and is normal on the right side. The Achilles-tendon reflex is prompt on each side. The right lower limb seems to be completely paralyzed. Pin-prick causes no movement of this limb, but probably causes some sensation of pain, as the man moves his left limbs as though he were in discomfort, and yet pain sensation appears to be diminished in the right lower limb. The Babinski reflex is not obtained on the right side, the toes not being moved in either direction; it is probably present, though somewhat uncertain on the left side." Leucocytes were 12,400.

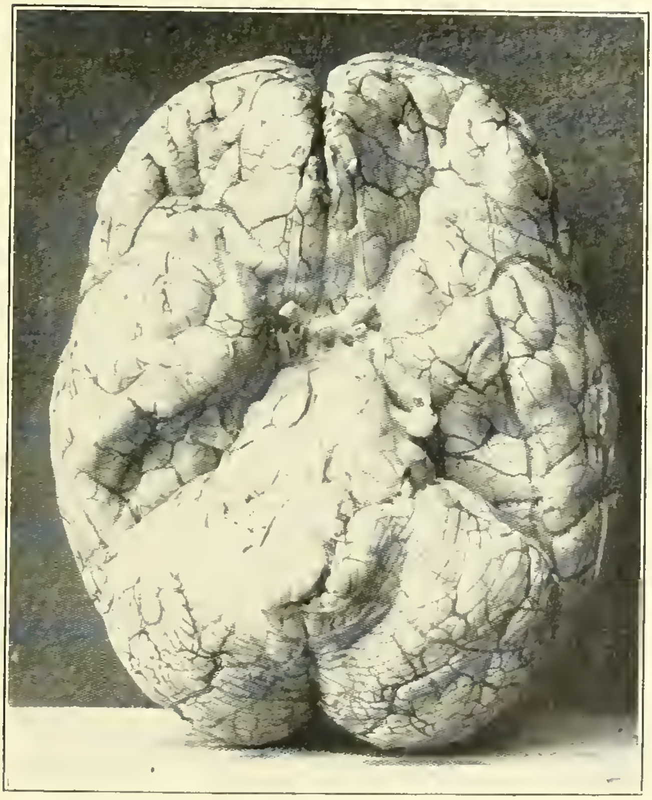

Fig. 3.—Case 3. Glioma occupying the entire pons.

Course of Disease.—The important features of this case were a gradually developing right hemiplegia, probably right hypalgesia, right hemianopsia, rigidity of the neck, a temperature that varied from 100 to 101, and the absence of ocular changes. Dr. Martin performed an exploratory operation April 28. Nothing abnormal was found. The right upper limb moved in response to electrical stimulation in the motor area. The man died April 30.

Autopsy.—A tumor occupied the greater part of the lower half of the left occipital lobe. It extended almost to the inner capsule and had the appearance of an angioglioma.

Case 3.—*Patient.*—Mrs. Y., aged 45 (Lab. No. 449), was seen by me March 23, 1908, in consultation with Dr. J. Thorington, by whose courtesy I report the case. Dr. Thorington sent the patient to me with the diagnosis of brain tumor, a diagnosis that seemed to me correct after an examination. My notes as taken from my case book are as follows:

General Examination.—"Mrs. Y. . . . has been complaining of vertigo about one year. When in bed she is obliged to lie on the right side; when she turns on the left side she feels 'sick and as though everything were going from her.' Her weight has decreased from 155 pounds to 121 pounds. Turning of the head to the left while lying down produces nausea. She vomits often in the morning. She has had pain in the muscles of the neck, and often a throbbing sensation in the frontal and occipital regions, especially when bending over. She is hungry all the time, and has a 'most vigorous appetite.' The menses are regular. The throbbing in the back of the neck is very severe and extends down the neck to the shoulders. She can not read unless the print is very large. She complains of kicking and jerking in her lower limbs, and can not lie down in the daytime because of this, and is kept awake at night by it. All symptoms began a little more than a year ago. The pain in the head is slight when she is sitting very still. She chokes when eating and swallowing, but if her attention is diverted she has no difficulty of this kind. She has three children, all healthy, and has not had any miscarriages. She complains of numbness all over both sides of the face, the numbness began on the left side of the face. The roof of the mouth also is numb. She has not had any pain in the face. She staggers on the street like a drunken person. Her behavior at times appears childish. The right pupil is a little larger than the left and both react to light, accommodation and convergence. She has marked nystagmus in looking to the right, and less in looking to the left, none in looking upward or downward. The nasolabial fold is slightly less distinct on the right side, when the face is at rest, but in showing the teeth or in drawing up either corner of the mouth separately, there is weakness of the left side in the whole distribution of the facial nerve. The right masseter is stronger than the left. Touch and pain sensations are normal in each side of the face. She has marked diminution of the conjunctival and corneal reflexes on the left side. She hears the voice and watch well on each side. The tongue when protruded sometimes deviates to the right, sometimes to the left, or is in the median line. The soft palate moves normally. The finger-to-nose test shows very little if any ataxia. The grasp of each hand is good. There is no impairment of voluntary movements in either upper limb. The biceps and triceps reflexes are exaggerated in each upper limb. Touch and pain sensations are normal in the hands and feet. Voluntary power is normal in the lower limbs. The patellar reflex is exaggerated on each side. The Achilles reflex is a little exaggerated on each side and about equally. There seems to be a slight Babinski reflex on each side, but it is uncertain. She has no ankle clonus. She sways very distinctly with the eyes

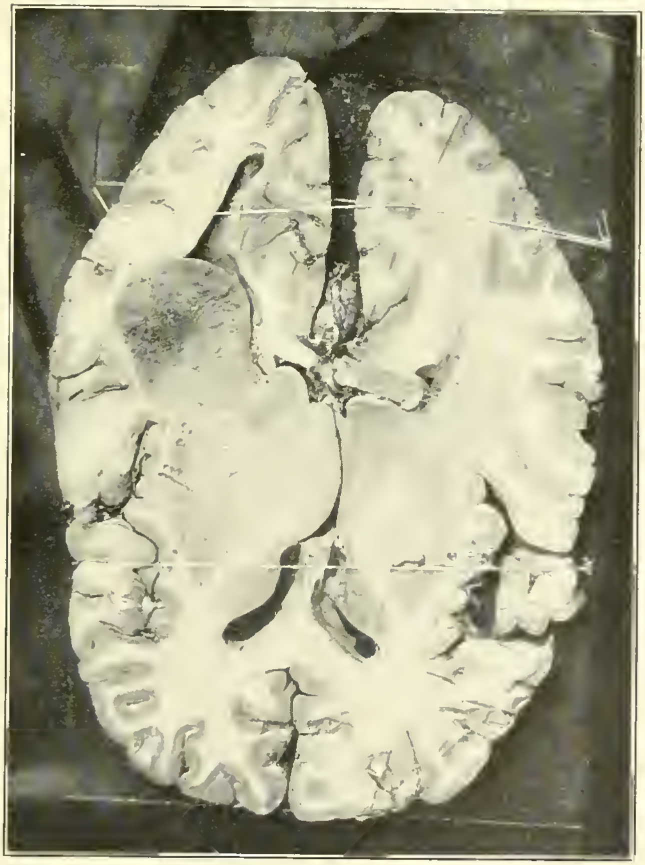

Fig. 4.—Case 4 Glioma in the region posterior to the right optic thalamus.

open, and more so with the eyes closed, and during my examination she falls frequently toward the right. The gait is ataxic, and more so with the eyes closed." She presented on one occasion adiadokokinesis in the right hand.

Ocular Examinations.—A letter from Dr. J. Thorington, dated March 21, 1903, stated: "The ophthalmoscope does not reveal any pathologic changes at this time. The eyegrounds are apparently normal and the discs are not elevated. Vision in each eye is normal, and the refraction + 1.25 sphere, together with presbyopia." On April 7, 1908, Dr. Thorington wrote: "Yesterday and today I noticed that the nasal side of each disc has commenced to be elevated and indistinct in outline; the retinal veins are enlarging and becoming tortuous." Dr. Thorington reported that the swelling of the discs reached 7 D. on April 13 and 10 D. on April 18. Operation was considered, but as nothing more than palliation could be offered the relatives declined to have an operation. Death occurred May 4, 1908.

Autopsy.—A glioma occupied the whole of the pons. The left optic thalamus and adjacent region were much larger than the corresponding area of the right hemisphere. The lateral ventricles, especially the left, were distended. A microscopic section from the tumor of the pons stained by the Weigert method, showed few nerve fibers. The anterior pyramids were well stained by the same method.

Case 4.—*Patient.*—D. T. (Lab. No. 422) was admitted to the Philadelphia General Hospital July 12, 1907.

History.—The patient had used alcohol, absinthe and tobacco to excess, but denied having contracted any venereal disease. He had been a clerk for twenty-five years. His symptoms began about ten months before his admission. He suddenly lost consciousness about thirty minutes, and six days later he had a similar attack which lasted three days. He could not describe these attacks more fully, but stated that he had had only two. He had had constant headache since the beginning of his symptoms, and it had varied in intensity, and was confined to the frontal region. He had been gradually losing power in his right upper and lower limbs, and his general vigor was impaired; he had lost his appetite and was unable to use his mental faculties as well as formerly.

First Examination.—His gait on admission was slightly hemiplegic, and he dragged the right toes. Sway was increased by closing the eyes. The pupils were unequal, the left being slightly larger. Response to light and convergence was preserved. There appeared to be some weakness of the right side of the face in showing the teeth. Both upper limbs were weak, the right more so. There was slight tremor in both hands. The biceps and triceps reflexes were prompt on both sides. The right patellar reflex was slightly exaggerated, and ankle clonus was present on the right side. The speech was slurring, and test words were pronounced with some difficulty.

His memory was poor, and he was easily confused. Soon after he entered the hospital he acted queerly, was dazed, attempted to disrobe in public, and did not know how to put on his clothing.

Second Examination.—Dr. Weisenburg, examining the man Sept. 18, 1907, noted that the right lower limb was contracted, and that the right upper limb was spastic. Right partial hemianopsia, and right hemihypesthesia were present.

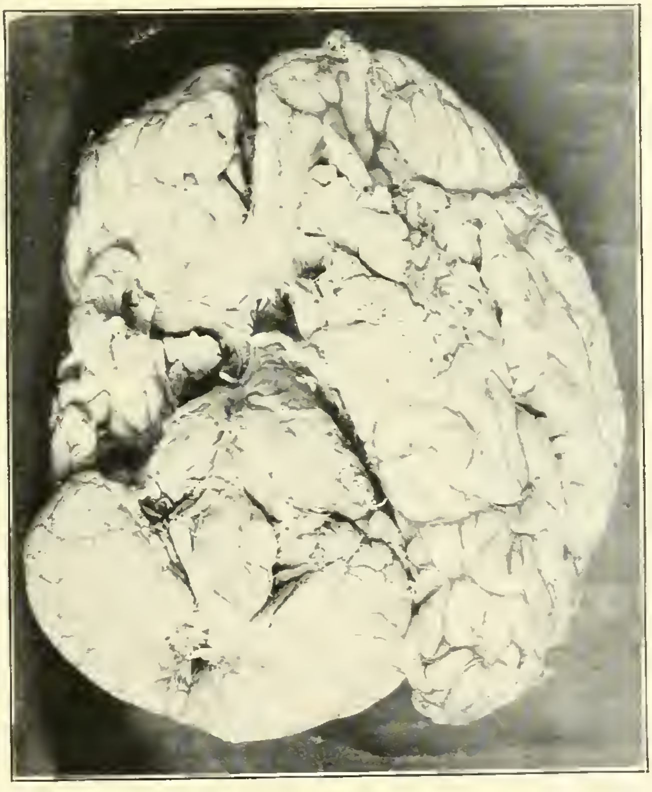

Fig. 5.—Case 5. Glioma of the left half of the pons and left half of the medulla oblongata.

Third Examination.—The man was examined by me Jan. 1, 1908. He was very stuporous, talked voluntarily, but irrationally. He heard when spoken to, but did not reply correctly. He had rigidity of the neck. The upper limbs were very rigid, especially the left. The upper limbs were adducted on the chest, with the forearms flexed on the arms, the left more than the right. The left palpebral fissure was larger than the right. The nasolabial fold was smoothed out on the left side.

The biceps and triceps reflexes were restrained by the spasticity of the limbs. Although there was no distinct conjugate deviation, he had a tendency to turn his head to the right, but the eyeballs did not deviate. The legs were kept flexed on the thighs, the right as much as the left. They could be extended to an obtuse angle with the thighs. The spasticity was great and equal in the two lower limbs. Ankle clonus was persistent on both sides. Involuntary twitching occurred in the lower limbs.

Course of Disease.—At the time he came under my observation the symptom-complex was bilateral, and as much pronounced on one side as on the other. The history stated that the hemiplegia had developed gradually, but the left side seems soon to have become affected. Unfortunately no examination of the eyes was made; at least no record was made of any examination. It probably would not have revealed papilledema. The condition became gradually worse and the man died Feb. 2, 1908.

Autopsy.—The right optic thalamus was much larger than the left, and the enlargement was uniform. The left was concave slightly on its inner side to accommodate the enlarged right optic thalamus. The tissue posterior to the right optic thalamus had distinctly the appearance of a glioma, and the tumor was fairly well defined, especially where it projected as a rounded mass into the posterior horn of the lateral ventricle. No limitation could be placed to the anterior border of the tumor.

Case 5.—*Patient.*—R. F. (Lab. No. 452) came into the University Hospital, July 23, 1908, referred by Dr. Rice. His age was 25. The notes taken at that time are as follows:

History.—"About nine months ago he began to have trouble with his eyes, his vision appearing indistinct and blurred. The disturbance has increased and for three or four months he has been seing double. The eyes ache a great deal. Shortly after the beginning of the visual disturbance he noticed that his lower limbs were unsteady, and the difficulty in walking has increased. Walking in the dark is especially difficult. During the winter he began to have severe headaches. At first these were daily, but they have become less frequent, until now he has them every three or four days. The severity of the headache has increased. The headache usually begins in the morning and lasts most of the day. He frequently vomits when he has a headache. The vomiting is not projectile, and he is nauseated when he vomits. Two or three months ago his speech became thick. He is dizzy when he has a headache if he attempts to move about. He is becoming a little deaf. He has never had convulsions, but a few times he has had a shaking of the left forearm. He has had some difficulty in passing the urine, but no disturbances of the bowels. He has not had any numbness. He has lost flesh. He denies venereal disease."

Examination.—This, made by me July 27, 1908, gave the following results: "In walking he staggers with the eyes open or closed, and the tendency seems to be to go more to the left. His face has a smoothed-out appearance, like the face in paresis. He wrinkles his forehead well and about equally on the two sides, although possibly there may be a little weakness in the upper distribution of the seventh nerve of the left side. The left eyelids can be opened more easily when closed in resisting passive motion; the left corner of the mouth is a little weakened both in showing the teeth and in drawing up the corners of the mouth separately. The tongue is protruded slightly toward the right. He has slight lateral nystagmus in looking to the extreme right or left. The hearing of the voice and of the watch is distinctly impaired in the left ear, but the hearing may be imperfect also in the right ear. The left side of the face in the region of the temporal and masseter muscles is flattened as though these muscles were much atrophied. In biting there is no contraction in either the left temporal or left masseter muscle, and the man says that his teeth do not touch on the left side. The left soft palate is paretic. Tactile and pain sensations are diminished in the distribution of the left fifth nerve, but not lost. The left conjunctiva and cornea are almost anesthetic. The grasp of the right hand is about equal to that of the left, and is fair. The biceps and triceps

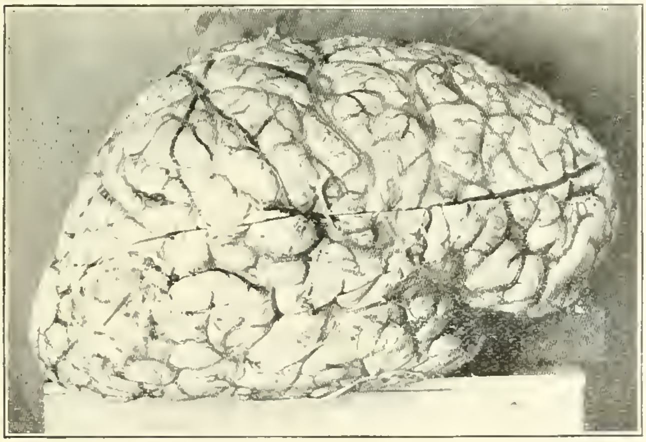

Fig. 6.—Case 6. Glioma of the right occipital lobe, indicated by a line.

reflexes are about equal, possibly a little prompter on the right side. Tactile and pain sensations are probably a little diminished in the left hand, but this is questionable. The pateller reflexes are about normal. He has a slight Romberg sign. The Achilles-tendon reflex is prompt on each side, and about equal on the two sides. The voluntary power in the lower limbs is about normal." The patient stated at this examination that he had had a "touch-like" sensation in the left side of the face, but only when he touched the face, and he had never had any pain in the face. On another occasion it was stated that the right pupil was slightly larger than the left, both eyes reacted to light and in accommodation, but the response seemed to be slower in the right eye. In testing taste there seemed to be no impairment to sweet or bitter substances. The recognition of objects in the hands and the sense of position were intact. A report from the eye dispensary stated that there was beginning papillitis, more advanced in the right eye. The fields were concentrically contracted. The left external rectus was paretic. The reaction of the left seventh nerve as tested by Dr. McConnell was normal to the galvanic current. The left fifth showed a quantitative but no qualitative change. The left side of the larynx was found to move less freely than the right side. The deafness of the left ear was attributed to catarrh, and Dr. Butler, in Dr. Randall's clinic, was unable to find any signs of nerve deafness.

An operation was performed by Dr. Frazier in the hope that a tumor in the region of the Gasserian ganglion might be found, although such a finding might be doubtful. Decomposition might be of benefit, and save the sight. It was the only chance offered to the patient for relief, and proved to be ineffectual.

Autopsy.—A tumor occupied the entire left half of the pons, the right half appearing normal to the naked eye. It involved the entire left half of the medulla oblongata, causing much enlargement corresponding to the great increase in size of the left half of the pons. The right half of the medulla oblongata appeared to be normal to the naked eye. The left cerebral peduncle was not enlarged. No tumor was found in the left Gasserian ganglion, and this ganglion seemed to be in good condition. A microscopic section of the medulla oblongata showed that the tumor invaded the right side. Nerve fibers were found in all parts of the section, but the tissue was rarefied as though the nerve fibers were pushed apart by the infiltrating glioma.

CASE 6.—*Patient.*—G. D. (Lab. No. 450), aged 50, was a patient of Dr. W. H. Teller, with whom I saw the case in consultation and by whose courtesy I report it. Syphilis was denied. The notes obtained March 18, 1908, are as follows:

History.—"Fifteen months previously while sitting on the porch he suddenly became aware of some visual disturbance, but was not unconscious, he could not see plainly, could not hold a pencil properly, could not say the words he desired to say; he was able to walk, he vomited, had severe pain over the entire head, slept badly the following night, but the next day he returned to work. He has not had any vomiting since that time. During the past three weeks he has seen objects before his eyes resembling snakes."

First General Examination.—"Pupils are equal; the reaction to light and in convergence is preserved; there is no nystagmus; no disturbance of tactile or pain sensation in the face. The patient can wrinkle the forehead, close the eyelids and draw up the corners of the mouth well on each side; the tongue is protruded in the median line; he hears the voice distinctly in each ear. The finger-to-nose test shows no incoordination; the power is good in both upper extremities, as is also sensation for touch and pain. The patient recognizes objects in either hand with the eyes closed, and has no impairment of the sense of position. The triceps and biceps reflexes are normal. The station and gait are normal with eyes open or closed. The patellar tendon reflexes are prompter than normal. The Achilles-tendon reflexes are prompt. Babinski's sign is not obtained. Sensation in the lower limbs is normal."

Second General Examination.—Notes taken on April 23, 1908, are as follows: "The right external rectus is weak. There is no hemianopsia, no anesthesia of the conjunctiva, no weakness in the facial distribution of either side. Touch and pain sensations are normal in the face. Masseter and temporal muscles contract well on each side. Tongue is normal. Hearing of voice and watch is good in each ear. There is no area of tenderness of the scalp, no stiffness of the neck. The power of each upper limb is good. A slight Romberg sign is obtained at this examination. He has had frequent attacks in which wriggling bright lights, beginning always in the right field, gradually passed to the center of the field, and disappeared in the left half of the field; these attacks were with dimness of vision, beginning also in the right field and disappearing in the left field. Since he has had optic neuritis he has seen black objects like snakes, rosettes, and forms of leaves in front of the eyes, and sometimes he sees the heads of persons." In other respects the examination gave much the same results as obtained at the previous examination.

Ocular Examination.—Iridic reflex to light is slow, prompt in convergence. There is limitation in outward excursion in both eyes, especially to the right. No limitation in upward movement. Pupils: O. D. 2½ mm. O. S. 2¾ mm. Vision O. D. 10/20. O. S. 10/40. Marked papilledema, periphery 2D., center 7D. No hemianopsia. Fields are greatly contracted.

Operation.—Dr. Teller operated. as we hoped to find a tumor in the left occipital lobe, or at least produce a decompression that would benefit the patient. No tumor was found at the operation.

Autopsy.—A tumor measuring 3 cm. in length and 2 cm. from above downward was found in the lower part of the right occipital lobe. Microscopically it had the appearance of a glioma.

I am indebted to Dr. Alfred Reginald Allen for the photographs.

4409 Pine Street.

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SYMPTOMATOLOGY AND LOCALIZATION OF BRAIN TUMOR*

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The most important indication of brain tumor is a gradual development of signs pointing to a sharply limited lesion of the brain; this may occur when the general symptoms of tumor, headache, nausea, vomiting and papilledema, are absent. It may, of course, be produced by other lesions, but in none is it so common as in tumor.

It is desirable to call attention in this connection to a symptom-complex that I have treated more at length in a paper read at the last meeting of the American Medical Association, namely, gradually developing hemiplegia. The importance of this complex is frequently overlooked. Since the presentation of the paper referred to I have had the opportunity of observing in consultation another case in which the only important signs of brain tumor had been a hemiplegia of uncertain beginning with hemianesthesia, in which, without any sudden increase of symptoms, the weakness had imperceptibly from day to day increased to a complete unilateral paralysis. Hemianopsia probably existed on the side of the hemiplegia. The diagnosis of hemorrhage or thrombosis had been entertained, and, as the patient lived in the country, an ophthalmoscopic examination had not been made. The very positive statement as to the slowness in the development of the hemiplegia led me to suspect brain tumor and, as the patient was a woman, to examine the breasts. A hard nodule with a retracted nipple was found, and further investigation elicited the information that the patient had had headache for some time, and other members of her family had had carcinoma. A necropsy was refused, and yet I do not feel venturesome in making a diagnosis of metastatic carcinoma of the brain. In this case is shown the importance of focal symptoms without the general symptoms of tumor. It

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* Read by invitation before the Chicago Medical Society, Oct. 27, 1909.

* From the Department of Neurology and the Laboratory of Neuropathology of the University of Pennsylvania.

We hear from the surgeons chiefly, but it is a truth that others also should emphasize, that operation in cases of brain tumor must not be delayed long—that to be successful it must be done early. There are two sides is not a bad idea to examine the breasts of a female patient who has symptoms of cerebral or spinal tumor. to this question, as to many others, and the neurologist more than the surgeon sees the early clinical aspect. A patient does not usually consult a surgeon for some cerebral lesion until he has been at least partially convinced that operation may be advisable. He may not have acknowledged this to himself, but his attitude toward operation is a receptive one. The neurologist more frequently sees the first indications of what may be brain tumor, and yet with his experience he often is unable to make a positive or even a probable diagnosis of tumor in an early stage of the disease. He must not be hasty or he will have many a head opened when there is no need. His responsibility is great. A necessity for moderate delay for observation and antisyphilitic treatment is not to be ignored, but the delay must not be too long or antisyphilitic treatment pushed too far. It must also be borne in mind that in glioma the symptoms may be decidedly lessened by the administration of an iodid, as in a recent case under my observation. The Wassermann reaction has become an important aid to diagnosis, but we must not forget that a syphilitic person may have a non-syphilitic tumor.

Especially serious is the delaying of decompression when papilledema is developing rapidly, as it may in tumor of the pons or cerebellum. I have known it to reach a high degree within a few days. As tumors of the pons often exist for a long time without causing papilledema, and then suddenly may give rise to a rapid swelling of the optic discs, the danger of being caught off one's guard is by no means imaginary.

As an illustration of the difficulties in deciding from the early symptoms as to the existence of a tumor a recent case under my observation may be briefly referred to.

A man of much intelligence with a syphilitic history had a convulsive attack confined to the left side of his body about a year ago. This was the only attack he had had. Headache and vertigo had occurred, but had not been important. No changes in the eyegrounds were found. A short time ago left hemiplegia developed gradually within a period of four days, and then gradually diminished and disappeared in a few weeks. These were the only symptoms, and yet they were suggestive of a focal lesion in the right motor region. Operation did not seem to me advisable at this early period, as the case was probably one of vascular disease. I was, however, prepared for the possibility of an operation later.

I am not in sympathy with the teaching that when there is any doubt as regards the existence of a brain tumor decompression should be performed and later the antisyphilitic treatment should be carried out. In my opinion, decompression is indicated in cases in which grave symptoms of increased intracranial pressure exist, and if the symptoms do not call for immediate decompression. I much prefer the antisyphilitic treatment before the skull is opened. Of course, the delay must not be too great, but the wise physician must learn by experience how long he dare wait. Decompression is a trauma of the head, and when mental failure has begun it may be intensified by this operation, as has occurred in my own practice, and in some instances decompression does not benefit the patient.

The interpretation of symptoms of brain tumor is not always easy. General symptoms may be mistaken for focal symptoms, and this is especially liable to occur if the supposedly focal symptoms appear late. The early appearance of focal symptoms has much greater diagnostic value than the appearance of the same symptoms after intracranial pressure has been increased for some time. No one makes a focal diagnosis from papilledema; at most he employs it only as indicating the side of the brain affected, but nerve deafness occurring with other signs of tumor has been regarded by almost all as of focalizing value. If it develop late in the symptom-complex there is a possibility that it may be merely a sign of intracranial pressure, and comparable with papilledema. Some evidence is at hand to indicate that possibly early appearing nerve deafness may be from general intracranial pressure, as in a case reported recently by Ruckert, in which tinnitus aurium and nerve deafness were among the first signs of tumor of the frontal lobes. Internal hydrocephalus may be used to explain certain cases of this character, or pressure driving the posterior part of the brain into the foramen magnum, but such lesions are not early even if the symptoms so produced appear to be early, and neither condition mentioned was present in Ruckert's case. A similar case has been reported by Souques, and explained as the result of general intracranial pressure. It is well perhaps to be a little cautious in regarding nerve deafness

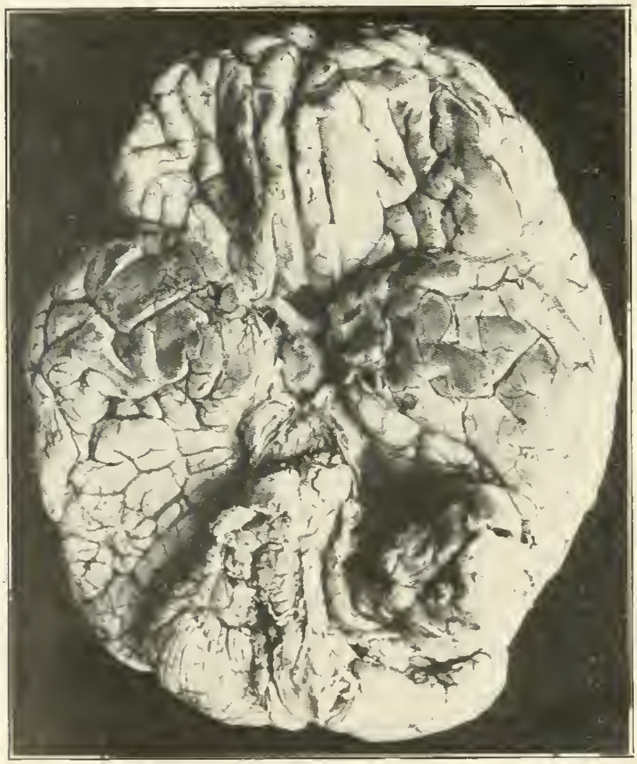

Fig. 1. Tumor which developed just below the tentorium and caused vertigo, ataxic gait, disturbance of speech, deafness, nystagmus, paralysis of the left external rectus muscle, some facial palsy, etc.

as one of the general symptoms of intracranial pressure; it is far more frequently a focal symptom, and not entirely parallel with papilledema.

Other cranial nerves may be affected by general intracranial pressure, but the signs of such involvement are usually late in appearing in the symptom-complex. I have known loss of smell to occur from a tumor at the foramen magnum, and paralysis of an external rectus muscle from a tumor of the parietal lobe. The abducens

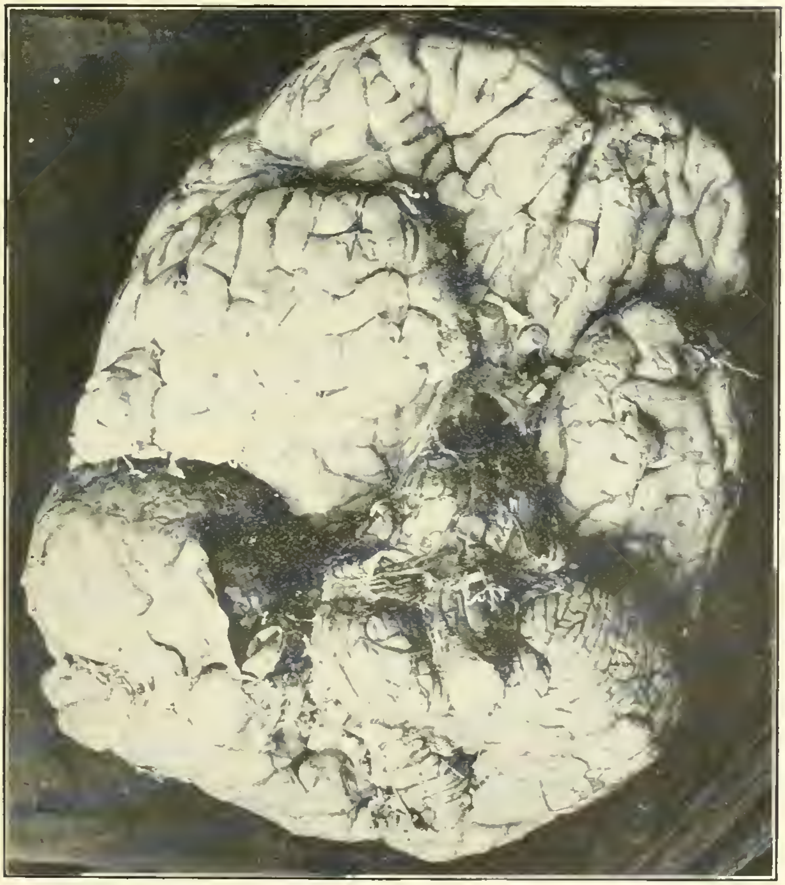

Fig. 2.—Tumor much larger than that shown in Figure 1, but growing above the tentorium. The only important signs were large head, nystagmus, papilledema, and diminished patellar reflexes.

nerve has the longest intracranial course of any of the cranial nerves, arising, as it does, at the union of the pons and medulla oblongata and leaving the skull through the sphenoidal fissure; and one should be cautious in accepting its paralysis as a localizing sign, unless when it occurs early.

Every experienced neurologist has met apparent contradictions between focal lesions and symptoms. Certain areas of the brain have been generally recognized as having to do with certain functions, and yet these areas have been destroyed without a corresponding loss of function. Various explanations have been sought. The statement has been made that a portion of the area must have been left intact, or the corresponding area in the opposite hemisphere must have assumed a vicarious function. These explanations have not satisfied all investigators;

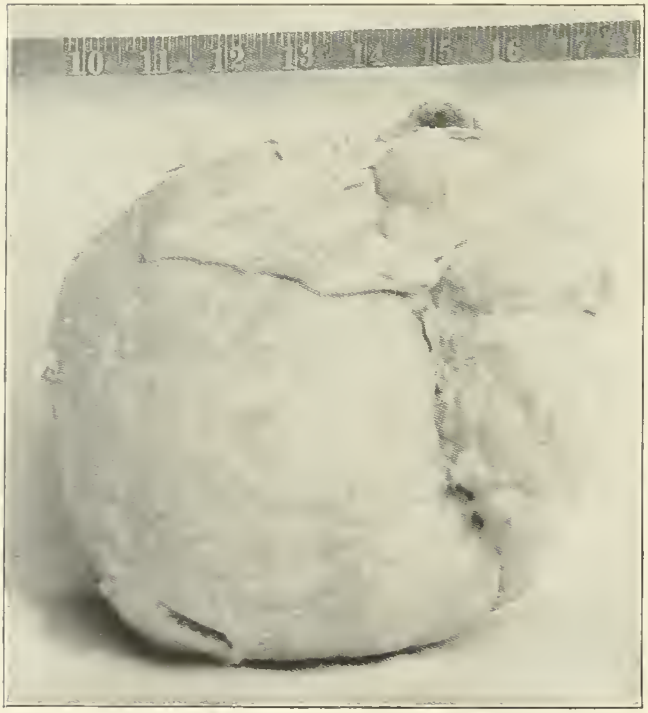

Fig. 3.—A larger view of the same tumor shown in Figure 2.

von Monakow has put forward his diaschisis theory, and insists that we must not identify localization of function with localization of symptoms. Localization of focal symptoms is a complicated reaction of the uninjured nervous system to the local cortical insult; whereas the localization of function depends on the action of all the elements of the function concerned in the entire central nervous system. When an area of the brain is destroyed, many tracts in connection with this area, commissural, associative, projectile, are injured or destroyed, and the effect is demonstrated by the disturbed action of distant parts of the central nervous system. The loss of tendon reflexes of the lower limbs in hemiplegia, for example, is the effect of diaschisis, as shown by disturbed function in the lumbar portion of the cord from a lesion of the cerebrum. As the various tracts differ to some extent in different individuals, the symptoms of a lesion, i. e., the reaction of the nervous tissue not directly affected in the lesion, varies from case to case, both in intensity and duration. We probably have in diaschisis one of the most satisfactory explanations for the apparent contradictions between symptoms and lesions that we frequently observe, and by it possibly we may explain astereognosis and other sensory disturbances following operations confined to the area of the

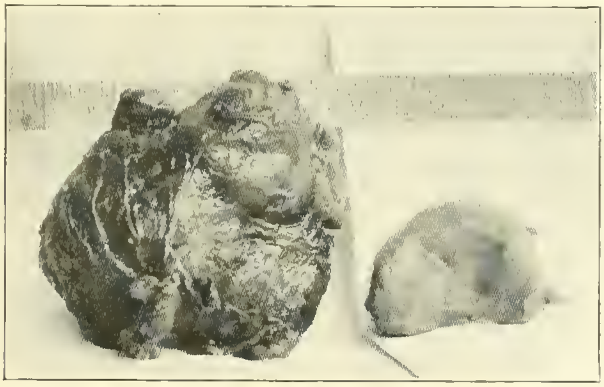

Fig. 4.—The larger of the two tumors here shown grew from the petrous portion of the right temporal bone and caused as first signs, twitching of the right side of the face and pain in the region of the ear. The smaller tumor was removed from the front part of the cerebrum, and was a glioma. Sudden death occurred five or six hours after the removal of the tumor.

brain anterior to the central fissure. In such operations, however, we must not discount the effect of exposure of a considerable area of the brain, of the results of handling the brain during operation, and especially the interference with the circulation of adjoining regions caused by the removal of a tumor or merely the ligation of vessels. The importance of the last procedure in causing symptoms has been demonstrated by the ligation of cortical vessels in the treatment of epilepsy.

In this connection I would refer to the recent valuable Linacre lecture by Sir Victor Horsley on the function of the so-called motor area of the brain. He describes a case in which, because of the athetoid movements of the left hand and later violent convulsive movements of the whole left upper limb, he removed the entire motor region for the upper limb in the gyrus precentralis, as determined by the response to electric stimulation. The whole depth of this gyrus was excised without any injury to the neighboring gyri or even to the vessels of the sulci, beyond the laceration of the smallest branches entering the portion of gyrus removed. Although not germane to the question under discussion here, it may be mentioned that the spasmodic movements totally disappeared from the moment that the gyrus was removed, and have remained absent to the time of publication of

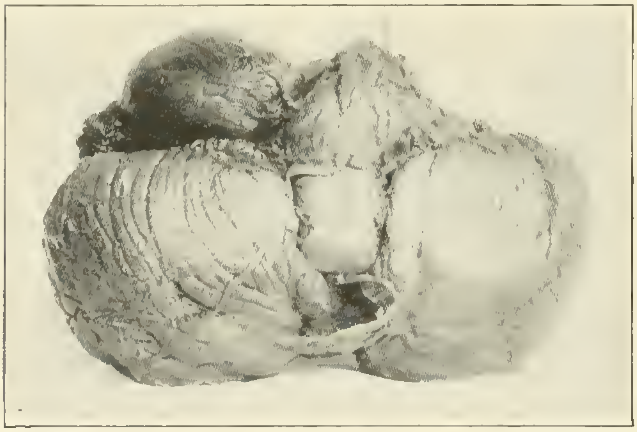

Fig. 5. Thrombosis of the inferior petrosal and cavernous sinuses, causing symptoms like those of tumor, lasting about a year.

the paper (thirteen months), and a month after the operation voluntary movements began to return and are increasing in efficiency.

The result of this operation led Horsley[1] to conclude that in man the gyrus precentralis is the seat of representation of tactility, topognosis, muscular sense, arthric sense, stereognosis, pain and movements.

We may, perhaps, ask whether this case proves all that is claimed for it. Granting that the motor area of the gyrus precentralis was accurately defined, and that it is possible to do so by electricity, concerning which I have some doubt, as electrical response may not be identical with voluntary motion; still we must all admit that after the excision of so large a piece of cortex, measuring approximately 4.5 cm. in length—if I read the somewhat indistinct photograph of the scale correctly—the disturbance to connecting tracts must be considerable, and the results of diaschisis so produced might well persist thirteen months. Therefore it seems questionable whether one may make so decisive arguments from this case for the sensory area of the gyrus precentralis. I have never been able to convince myself, however, that the gyrus precentralis is entirely motor and the gyrus postcentralis entirely sensory. From *a priori* reasoning this conclusion seems improbable. The regions of the brain are not separated so sharply from one another as to form a map with outlines as definite as those of the United States, and we all admit that the motor cortical centers are not sharply defined from one another. It seems decidedly schematic to say that the central fissure is a line of sharp definition—indeed, the bottom of the fissure is supposed to be the line of definition—that in front of it is the entire motor area, and behind it the entire sensory area. The same objections to these conclusions in this extremely valuable paper of Horsley's may be made to some of those in Hoppe's recent important paper[2] on the location of stereognosis in the gyrus precentralis.

Possibly more valuable in determining the representation of sensation in the precentral region is the experience of Cushing,[3] who in a person not under the effect of an anesthetic obtained the sense of muscular movement from the gyrus precentralis by electrical irritation. He states that his patient was perfectly aware of the movements, and described his appreciation of them as a drawing sensation, as when the muscles are "pulled" by peripheral electrical stimulation. Was this, however, the stimulation from the muscular movement conveyed from the limb through sensory fibers to the parietal lobe?

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[1] Horsley: Brit. Med. Jour., July 17, 1909, p. 125.
[2] Hoppe: Jour. Nerv. and Ment. Dis., September, 1909.
[3] Cushing: Brain, 1909, xxxii, 48.

Horsley's case seems to show what he claims for it regarding motion, namely, that motion is not represented solely in the gyrus precentralis, and to justify his conclusion that the gyrus postcentralis is in man part of the area for the upper limb in which the sensorimotor representation is of the same kind as that in the gyrus precentralis, but in it probably provision for sensorial coordination is greater, and that for efferent impulses less. It is to be remembered that he destroyed the whole motor area for the upper limb in the gyrus precentralis and yet much voluntary motion returned. Horsley states that such conclusions were made probable by the experiments of Grünbaum and Sherrington on the chimpanzee, in which animal these observers found that after the arm area in the so-called motor precentral gyrus was excised the purposive movements returned in a few weeks, and compensation was not effected by the opposite arm area in the contralateral hemisphere.

Horsley shows by his case that the Betz cells are not necessary for motor function, and refers to the fact that Brodmann likewise demonstrated that they are not indispensable for purposive muscular actions; therefore conclusions based on the histology of the cortex in defining the motor region contain an element of uncertainty. The same is true of Campbell's conclusions as to the sensory area posterior to the Rolandic fissure as determined by the alteration in a sensory nervous disease as tabes, conclusions which have been shown to be erroneous by Gordon Holmes.[4]

In November, 1899, I reported[5] that I had employed the degeneration of amyotrophic lateral sclerosis extending to the cortex as a means of determining the extent of the motor cortex in man. No one previously had used this method, and yet the degeneration of this disease is chiefly one of the motor system. The gyrus postcentralis was not intact in my case, but was considerably less degenerated than the gyrus precentralis. It is true that tracts other than motor are sometimes slightly degenerated in amyotrophic lateral sclerosis, but this degeneration probably is not early and may be secondary. I am not aware that anyone has observed pronounced degeneration detectable by the Marchi stain in the posterior columns in this disease.

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[4] Holmes, Gordon: Rev. Neurol. and Psychiat., 1908, p. 5.

[5] Spiller: Jour. Nerv. and Ment. Dis., 1900, p. 165; Contrib. from William Pepper Lab. Clin. Med., 1900.

Later Campbell employed this means without reference to my work, but was unable to find degeneration in the gyrus postcentralis. Rossi and Roussy[6] still later found degeneration in the gyrus postcentralis by this method.

It is singular that a case very similar to Horsley's should be reported by P. L. Friedrich[7] about the same time that Horsley's paper appeared. The upper limb center in the gyrus precentralis was defined by electrical irritation and excised because of epilepsy. The return of motor power after five and a half weeks was very marked. Contrary to the findings in Horsley's case, sensation was not altered in any way. The operation produced a cure, at least until the time of the report, six months after the operation. Friedrich explains the return of power as the result of "compensation," and implies that it was from the opposite cerebral hemisphere, although this statement is not clearly made. Before this question of the method by which return of power occurs can be satisfactorily answered it will be necessary to have the results of excision of the centers for corresponding limbs in both cerebral hemispheres in man, and it is not likely that these will be obtained soon.

Few American or English surgeons or neurologists, so far as I know, have advocated cerebral puncture. As yet this procedure seems to be recommended chiefly by certain German physicians. It has the approval of Oppenheim, but is discountenanced by Krause, and has led to open letters between these two men. There are grave dangers in connection with it that have deterred many from adopting it, and it seems doubtful whether it will ever come into general use.

It is likewise doubtful whether the search for tumor cells in the cerebrospinal fluid by lumbar puncture will be of much service. It is true that Sicard and Gy[8] obtained sarcoma cells in this way in sarcomatous meningitis, but a solitary tumor would be unlikely to produce sarcoma cells in the cerebrospinal fluid, and sarcomatous meningitis is extremely rare. Lumbar puncture also is serious in cases of suspected tumor of the brain and has repeatedly caused a fatal termination.

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[6] Rossi and Roussy: Rev. Neurol., Aug. 15, 1907.

[7] Friedrich, P. L.: Monatsschr. f. Psychiat. u. Neurol., 1909, xxvi, 129.

When multiple fibromas occur in superficial parts of the body associated with signs of tumor of the cerebello-pontile angle, they are regarded as indicating that the cerebral tumor is likewise a fibroma. In none of the cases of tumor of the cerebello-pontile angle that I have seen were fibromas of superficial parts found, and this association of superficial and deep growths seems to be so rare as to be of comparatively little value in diagnosing a brain tumor, although occasionally it is of much importance, as in a case described to me recently by Dr. G. Bastianelli, of Rome, Italy.

The *x*-rays also have been unsatisfactory in my experience. I have never been convinced that the shadows shown and supposed to indicate brain tumors were reliable. When the bone of the skull is much thinned, as from increased intracranial pressure or when the sella turcica is enlarged, *x*-rays may be of service.

I have examined the specimens of brain tumor in my possession in the Laboratory of Neuropathology of the University of Pennsylvania, and have counted 72; probably some have been overlooked. By far the larger number of these (40) were in the cerebrum. The tumors in the pons (7), in the cerebello-pontile angle (8) and in the cerebellum (7) were nearly the same in number. Most of the tumors are gliomas or sarcomas, and the numbers of these two varieties differ according to the interpretation of the examiner. The diagnosis between glioma and sarcoma may be difficult, and the term gliosarcoma is an attempt to avoid the distinction.

The diagnosis of gliosarcoma usually implies a tumor that has resemblance to both glioma and sarcoma, and not usually a tumor partly a glioma and partly a sarcoma. Such a mixed tumor must be rare and difficult to recognize, but that it may occur is shown by the report of a recent case by Merzbacher.[9] In this a tumor of the brain contained as a center a sarcoma with a periphery of glioma. The sarcoma was considered the primary tumor, and Merzbacher speaks of glioma following sarcoma or reactive glioma in preference to gliosarcoma.

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[8] Sicard and Gy: Rev. Neurol., Dec. 15, 1908, p. 1245.

[9] Merzbacher: Monatsschr. f. Psychiat. u. Neurol., August, 1909, p. 176.

Only two gliomas in my collection were fairly sharply defined, and one of these was infiltrating at one portion. I have had no sharply defined glioma with one exception, and I am a little in doubt in regard to this. Well-defined gliomas are reported by others. I have never had any finding that would lead me to believe that a glioma may entirely disappear and give place to a cyst.

Dr. F. B. Mallory regards sarcoma as a rare tumor of the brain, and some tumors sharply defined that would be classed by many as sarcomas he places among the gliomas. The fibromas or fibrosarcomas within the cranium he classes as endotheliomas.

I would call attention especially to the small number of macroscopic gummas (4) in my collection. We often hear the diagnosis of gumma made when cerebral syphilis is suspected, and yet the findings do not justify this diagnosis. I have for years taught students to be careful in making the clinical diagnosis of gumma of the brain. Cerebral syphilis is extremely common, but the usual lesions are arteritis and meningitis, and it is because of this that antisyphilitic treatment may be so valuable in the early stages of syphilis of the nervous system before degeneration has occurred. I do not believe that it is possible to dissolve by drugs a well-organized growth.

The tumors growing in the pons (7) in my specimens were: glioma 4, tubercle 3. Tumors of the cerebello-pontile angle are almost always fibromas or fibrosarcomas, although by some these growths are described as endotheliomas. They differ from the tumors I have regarded as endotheliomas, which develop from the dura. The cerebello-pontile angle tumors are well defined and easily removable, so far as their attachments are concerned, but they are situated in such a dangerous region that the attempt to remove them is almost always fatal, or, if not fatal, the condition of the patient usually is not improved by the operation, or is even rendered more pitiable. It seems, indeed, strange that the most favorable form of tumor (fibroma) as regards structure and sharp definition should favor above all portions of the brain the cerebello-pontile angle, the most vital region.

Carcinoma rarely occurs; in my collection it is present in 3 cases; nor is tubercle very common. I have seldom found multiple tumors, and this infrequency is important as regards operation. Most tumors growing from the dura are fibromas, fibrosarcomas, or endotheliomas; occasionally gumma is found, and these are all favorable for operation. The gumma is more likely to be a superficial tumor, but is never sharply defined; when growing in the brain substance it is not very favorable for operation, but may be so when confined to the dura.

Sarcomatosis, or tumor meningitis resembling sarcoma or glioma tissue, I have seen four times. It seems to be little known in this country, and the clinical diagnosis because of the diffuseness of the symptoms, is extremely difficult. Where a sarcoma develops elsewhere in the body and is recognized, sarcomatosis of the nervous system also may be diagnosed, or it may be suspected where symptoms of brain tumor are associated with those indicating focal symptoms of the spinal cord.

Occasionally a condition of general clonic spasms occurs in tumor of the brain, resembling that of a patient in a state of acute alcoholism. It is not unlike the epilepsia partialis continua, to which I called attention in this country in June of this year,[10] although it has been known to Russian writers many years. I have recently had an opportunity to observe this general clonic spasm in a man who had a large tumor extending almost to the cortex and occupying the motor area of the brain. If such a case were brought before the physician without any knowledge of the previous history a diagnosis of alcoholism might be excusable.

The symptoms of tumor are seldom mistaken for the nephritis of pregnancy, but this is a possibility that must be considered. In one of my cases (No. 296) the early symptoms of tumor (an endothelioma) occurring during the late stage of pregnancy and persisting after delivery might have led to a diagnosis of nephritis of pregnancy. Papilledema, convulsions, nausea, vomiting, and headache might easily be explained by such a diagnosis. Unfortunately the report of the urinary examination has been lost, but an examination was certainly made. The loss of tendon reflexes of the lower limbs, paresis of the left facial nerve and of the left external rectus muscle which occurred in this case would suggest a focal lesion, but these symptoms were few and slight considering the large size and position of the tumor. The escape of the fifth nerve is noteworthy with a tumor situated as this was, near the cerebello-pontile angle, but above the tentorium. Cases of tumor without any focal symptoms are by no means rare, and one may readily understand the difficulty that might arise in diagnosing between nephritis of pregnancy and tumor.

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[10] Spiller: THE JOURNAL A. M. A., June 12, 1909, lii, 1921.

As compared with the case just mentioned, another case of tumor (a fibroma, No. 338) was typical, and yet the two tumors were in nearly similar situations. In the latter the symptoms were vertigo, ataxic gait, occipital headache, thick and slow speech, failure of hearing on the left side, and failure of sight with optic neuritis, slow nystagmoid movements in attempts at rotation, paralysis of the left external rectus muscle, cessation of menstruation for almost six months, slight weakness of the left side of the face and of the left muscles of mastication, exaggeration of patellar reflexes, especially of the right with patellar clonus, and uncertain Babinski reflex on the right side. The diagnosis of a tumor of the left cerebello-pontile angle was as certain as such a clinical diagnosis ever can be.

Still more illustrative of the difference in the symptomatology of a tumor immediately above the tentorium and, therefore, in the middle cranial fossa, and of one immediately below is the following case (a fibrosarcoma, case No. 337), in which the tumor was exceedingly large and measured 8 cm. in diameter, and was round as a ball, and yet the symptoms were indistinct. The man was a patient of Dr. Mills' and was for a time under my care. His father was said to have a very large head, of about the same size as the patient's. The man was admitted to the hospital Feb. 20, 1906. About five months previously he had what was diagnosed as acute Bright's disease. Then he complained of his eyes becoming weak so that he could not see so well as formerly, but he states that for some years he was obliged to squint in order to get a good view of an object. He had no pain, no nausea, no vomiting (except vomiting during a short period), and no vertigo. His only serious symptom was impairment of vision. The report from Dr. de Schweinitz concerning the eyes was:

> Vision, O.D. 6/100; O.S. 6/100. Horizontal nystagmus, ocular movements free. Convergence practically nil, O.S. deviating. O.S. shows blurring and striation of nasal side of disc with some dipping of vessels, beginning neuritis +3D.,

large crescent out but edges seen. The swelling in O.D. is very vascular. O.D. shows a decided choked disc, height 10-11 D.

His head was so large as to suggest hydrocephalus. In looking directly forward he had lateral nystagmus and also occasionally in looking to either side; it seemed to be more an ataxic movement of the eyeballs. Finger-to-nose test showed slight ataxia on each side. Circumference of the head measured 25 inches, and from one external auditory meatus to the other 14 inches. The patient wore a 7¾ hat. The patellar reflex was almost lost on each side. He did not have Romberg's sign. Gait with the eyes closed was a little ataxic, but he did not really stagger.

As the symptoms might be interpreted as those of tumor, although hydrocephalus was possible, he was referred to Dr. Frazier's service. Operation was performed February 21 over the occipital bone and a portion of the lateral lobe of the cerebellum was removed, but the patient's condition was so serious that the operation had to be hastily concluded. He died March 9.

The necropsy was performed by Dr. Lavenson. The brain without the tumor weighed 1,920 gm., with it 2 110 gm. The convolutions were considerably flattened. The tumor weighed 190 gm. and was found on the right side of the brain adherent to and apparently taking its origin from the tentorium cerebelli. It was entirely encapsulated and nowhere firmly adherent to the brain tissue. It had destroyed the anterior basal portion of the right occipital lobe and the greater portion of the basal part of the right temporal lobe. The tumor was firm, dense, and smooth. Two or three smaller nodules adherent to it were found at its attachment to the dura. A growth situated as this was at the base of the occipital lobe may not interfere with the optic radiations, which are situated higher, and therefore may not cause hemianopsia.

Vomiting was slight, and there was no headache and no vertigo. The only important signs were the large head, nystagmus, changes in the eyegrounds, and diminished patellar reflexes; truly very insufficient disturbance when the large tumor is taken into consideration. The case shows the extraordinary degree of pressure the brain will occasionally endure, and yet in most cases such a growth would cause grave symptoms.

The early appearance of symptoms in basal tumor probably depends on the early involvement of one or more cranial nerves in the growth. If these nerves are merely distorted by the developing tumor, especially if the distortion be of slow development, the symptoms are much less intense.

Another case (an endothelioma, No. 494) I may refer to in which the tumor developed in the posterior fossa and, although small, gave very definite symptoms. At first it was not easy to exclude entirely a lesion in the petrous portion of the temporal bone, or a multiple neuritis of cranial nerves. The insidious onset that a tumor may have was well shown in this case. The twitching of the right side of the face with pain in the region of the ear was the earliest and for some weeks the only sign of tumor. The subjective disturbance of hearing preceded many weeks any objective deafness, and this is one more evidence that subjective disturbances are likely to precede objective. A patient's statement of his symptoms, provided he be intelligent, may be of great value to us in diagnosis when our examination reveals nothing. In the recent case reported by Horsley and referred to above, the statement is made that several days before the movement of the fingers and thumb returned the patient stated that he felt that the power was returning, and that he would shortly move the members.

In my case considerable complaint was made of numbness in the right side of the face, although no impairment of sensation could be detected by examination. I have seen a subjective sense of weakness in a limb precede by twenty-four hours any objective paresis.

The diagnosis of a lesion, probably tumor, near the exit of the seventh and eighth nerves was not difficult when the pain and twitching of the right side of the face were followed by complete right facial palsy, right nerve deafness, paresis of the right side of the soft palate, some ataxia in walking, and exaggerated left patellar reflex with left ankle clonus; but for a long time it was very difficult to decide whether the lesion was in the inner ear or within the cranium, and at first the possibility of multiple neuritis of the cranial nerves had to be considered, and also that of glioma of the pons and medulla oblongata, as glioma does not produce symptoms from all the structures it implicates. The diagnosis of location was fully confirmed by the two operations that became necessary.

Both Dr. Frazier and I were very loath to have a second operation in this case, as the patient nearly died after the first, but he and his relatives insisted on having it performed, and it did not seem right to refuse him the one chance for his life. The tumor at the second operation was removed by Dr. Frazier.

It is worthy of note that no evidence of papilledema was present in this case, although the growth was in the posterior cranial fossa and of fairly large size. When removed it measured 4 cm. in length. The pain began in the region of the right ear, and was one of the earliest symptoms, and sometimes the ear and adjoining part of the face were tender to touch. The pain was not in the forehead unless it radiated there from the ear. The possibility of this pain being caused by irritation of the seventh nerve must be considered. It was not in the distribution of the fifth nerve, and there were neither objective sensory nor motor signs of implication of this nerve. It followed twitching of the right side of the face and, therefore, like the twitching, seemed to be caused by irritation of the facial nerve.

The twitching of the facial muscles from irritation of the facial nerve, occurring in attacks and resembling cortical irritation, is a phenomenon not very well known. It was present in this case, and I have known it to occur in at least two cases in which I was associated with Dr. Mills, who has put these cases on record.[11] The first case occurred about 1900 and was reported by Dr. Mills and me at that time before one of the Philadelphia medical societies. The spasms were witnessed by both of us, and was so suggestive of cortical irritation that it was difficult at that time, without any similar occurrence reported in the literature, to attribute it to irritation of the facial nerve. It differed from the more common forms of facial spasm in that it occurred at rather long intervals and was more like an epileptic attack. Further reference has been made to these cases by Dr. Weisenburg.

It was desirable in the case to which allusion has been made above to determine whether the tumor implicated the seventh and eighth nerves at their exit from the cranium or at their exit from the pons where it joins the medulla oblongata. The symptoms did not permit a differential diagnosis as regards these two locations, and I doubt whether such a diagnosis would be possible.

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[11] Mills: Boston Med. and Surg. Jour., April 26, 1906.

Sudden fatal termination is common in tumors situated anywhere in the brain, but especially when the tumor is in the posterior cranial fossa. Operation was attempted by Dr. Frazier in one of the cases referred to in this paper (No. 296), in which after an opening about the size of a silver dollar had been made in the occipital region the patient suddenly became pulseless and respiration ceased. Her condition improved, but she died the same day.

The explanation may be that the tumor in this situation causes dislocation of the posterior part of the brain into the foramen magnum, but a more probable explanation seems to me to be that the cardiac and respiratory centers of the medulla oblongata are damaged by the pressure of the tumor and are less likely to withstand an operation.

In another case (Rosenfeld) in which the diagnosis was cerebellar tumor, decompression over the cerebellum was performed. The patient, a child 8 years old, rallied from the operation and slept well during the following night. The next morning with the temperature 98 degrees, pulse 84, and respiration 20, she suddenly began to gasp for breath and then ceased breathing, and soon the heart ceased to beat. This sudden death was observed also in a cerebellar case without operation (Lokuta), and in a case of cerebellar tumor (McClain), after decompression over the posterior cranial fossa. The patient, a boy aged 13, did well after the operation by Dr. Frazier for ten days, and had been in a wheel chair frequently. On the tenth day about midday he suddenly became very cyanotic, respirations became rapidly reduced until they practically ceased, and he sank into profound stupor. After prompt and prolonged artificial respiration, administration of oxygen and stimulation, the cyanosis disappeared and the patient recovered from the attack. Near midnight of the same day he had another attack of respiratory failure with cyanosis similar to the previous one and gradually sank and died within two hours.

Sudden death is not confined to cerebellar lesions, as shown by still another case (No. 495) in which death occurred very suddenly five or six hours after the removal of a small tumor, a glioma, by Dr. Frazier from the front part of the cerebrum, when the patient had seemed to be in excellent condition for some hours after the operation until twenty minutes before death (Fig. 4). It would be easy to add other illustrative cases.

The causes of sudden death are numerous and not always detectable even at necropsy. Candler has shown[12] that hemorrhage into the suprarenal capsule may give rise to obscure abdominal symptoms or nervous manifestations difficult of diagnosis, and may even be a cause of death without any appreciable symptoms whatever, and there are many other ways in which death, even sudden death, may occur without the cause being clearly demonstrated. To one aspect of a case in which sudden death occurred (Lokuta) I would call especial attention. A Pole with symptoms of tumor of the posterior cranial fossa was evidently very much frightened when brought into the operation room, and went into collapse just before etherization was begun. The attack which preceded etherization seemed to be brought on by fear of the operation. It is the only instance in which I have known fear to have such serious results in brain tumor, and the occurrence seems to indicate that severe depressive emotion may impair the chances in a serious cerebral or possibly cerebellar operation, much as Crile has demonstrated it may in operations on the thyroid gland.

In this case, as in others I have tested, the conjunctival and corneal reflexes were normal. Oppenheim regards a diminution of these reflexes as indicative of cerebellar tumor, and when unilateral as being on the same side as the tumor. I have never found a loss of these reflexes in cerebellar tumor. I would suggest that where the test is made with a piece of cotton wool the examiner may be deceived, as many normal persons may stand this sort of touch of the eyeball without winking.

In one case (No. 338) the left motor fifth was affected, but the sensory portion of this nerve escaped. In another case (No. 477) the sensory portion of the fifth nerve was affected by pressure of a thrombotic inferior petrosal sinus and the motor portion escaped. We have in this one of the peculiar selective actions of lesions in producing symptoms so common in nervous diseases.

Ataxia in walking and standing may be increased by closing the eyes, notwithstanding the lesion is cerebellar.

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[12] Candler: Arch. Neurol and Psychiat., 1909, iv.

It is often stated that a diagnostic point between the ataxia of tabes and that of cerebellar disease is the absence of increase in the ataxia on closing the eyes in the latter disease. This is not an absolutely reliable sign, as in some cases of cerebellar ataxia closing the eyes increases the incoordination.

Repeated attacks of hemiplegia are a rare manifestation of tumor. In one of the cases (No. 495) left hemiplegia with stupor and convulsions followed by almost complete recovery, and later by a return of the palsy with stupor and convulsions, seemed to be a form of exhaustion paralysis. That paralysis may follow convulsions and last several days, and even much longer if the convulsions be repeated, is well known. There could not have been any sudden increase in the size of the tumor to cause two attacks of paralysis, nor could the condition be regarded as uremic. The exhaustion paralysis of epilepsy was strikingly shown in a case reported recently by Williams Cadwalader,[13] and the literature is given by him.

It is well to remember that a cyst of the cerebrum or cerebellum is usually in association with tumor, and it is proper, therefore, to search for tumor when a cyst is found at operation. In one case (No. 326) the diagnosis of cerebellar tumor was easy. The improvement after the finding and evacuation of a cyst was striking, but it became my painful duty when informed of the results by letter to suggest that caution was needed. I much feared that the evacuation of the cyst would produce only temporary relief by diminishing the pressure on the brain. Such proved to be the case, and a glioma of the cerebellum was found later. A cyst is very often associated with a glioma.

The important question as to whether Jacksonian epilepsy is a sufficient cause alone for operation on the brain is of importance. The Jacksonian spasm in a case (Davenport) observed by me was for about a year and a half the only sign of cerebral lesion, and it was not until after this period that slight motor aphasia and slight hemiparesis developed. Headache and papilledema were not symptoms. I felt that an exploration of the brain was desirable, because convulsions strictly unilateral occurring occasionally during eighteen months were very suggestive of focal lesion, and such a focal lesion most likely would be tumor. The existence of idiopathic unilateral epilepsy has been discussed, and such a condition is clearly recognized, even status hemiepilepticus of idiopathic character. Shall we or shall we not operate in such a case as that mentioned above? I am distinctly in favor of exposing the brain at the region indicated by the symptoms. The possibility that we may not find anything abnormal is not to be ignored, and yet the hope in operating on brain tumors lies in the removal of the tumors while they are small. The motor area responds promptly to the irritation of a small tumor that might remain latent in many other parts of the brain. If this recommendation be followed, operations will probably be performed without revealing tumors, but that is the less of two evils. I am well aware also that the mere opening of the skull may prove fatal, occasionally by infection of the brain, as when a patient in stupor disturbs the bandages; or by postoperative hemorrhage from a small vessel, or by injury of one of the large venous sinuses with extensive hemorrhage, or when a piece of bone is removed for decompression and occlusion of vessels in a cerebral hernia occurs with extensive cerebral softening, or, still more rarely, from some unknown or remote cause as pulmonary embolism. I have seen death produced in all these ways, and yet we must remember the extreme seriousness of brain tumor; that operation alone affords any hope of recovery or of prolonging life, unless perhaps in tumor of the pituitary body, which is often of slow growth; and that operation must be done early if it is to be successful. Therefore, when symptoms are persistent, indicate a focal lesion of the brain, and are gradually increasing, even though general symptoms of increased intracranial pressure are absent, such as papilledema, headache, nausea and vomiting, I believe that as a general rule operation is advisable if the lesion, as shown, for example, by Jacksonian epilepsy, gives any reasonable hope of being found near the surface of the brain. Palliative operation is not to be considered in such a case as I have described, because the occasional unilateral spasms, the slight aphasia and slight hemiparesis would not seem to warrant it. Decompression, in my opinion, is more serious than mere exposure of the brain with return of the osteoplastic flap to its proper position—more serious because from the removal of a portion of bone, the size or less than the size of a silver dollar, a hernia cerebri is very likely to occur, and sometimes the hernia becomes very large, even becomes infected and forms fungus cerebri, or by pressure of the brain against the bone and occlusion of vessels leads to extensive cerebral softening.

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[13] Cadwalader, Williams B.: The Journal A. M. A., Nov. 21, 1908, li, 1778.

In one of my cases of probable tumor of the pituitary body (G. S.) in which the most important symptoms were bitemporal hemianopsia, some mental dulness, tendency to jocosity, and marked asthenia, a peculiar sexual disturbance was present, first detected by Dr. Thompson S. Westcott, under whose care the patient had been. This patient had failure of ejaculation with preserved normal sexual desire and normal erection. It is well known that the genital functions in both male and female are impaired or lost as a result of lesion of the pituitary body, but the dissociation of the sexual functions, as manifested in this case, is unusual, and like that which sometimes occurs from lesions low in the spinal cord.

The diagnosis of frontal tumor is often difficult. In one of my recent cases I depended on the jocosity (*Witzelzucht*) and apathy, in connection with general symptoms in diagnosing frontal tumor, and the tumor was found in the frontal lobe; when, however, we try to decide between the right and left frontal lobes we often are helpless. The patient to whom I refer (R.) had come for the purpose of operation from a distant city. He informed me when I asked for his hand in examination that he had four hands, and this he said as a sort of joke. Such a statement as this under such serious circumstances indicates a peculiar condition of the brain, and probably a frontal lesion.

In one case only have I seen a pressure palsy of the seventh nerve follow operation, and in this it was caused by the head-rest and was of short duration. The facial nerve is not likely to be affected by any pressure from without, but pressure palsy of it has been reported. Knapp, in a case reported by him,[14] found that it resulted from sleeping a couple of hours with the cheek on the table. When the man awoke the face was paralyzed on one side from the pressure, and the faradic and galvanic reactions of this side were quantitatively diminished. Recovery occurred within about four weeks.

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[14] Knapp: Monatsschr. f. Psychiat. u. Neurol., October, 1905, p. 309.

The symptoms of brain tumor may be caused by a variety of lesions, especially well recognized in this connection are brain abscess, hydrocephalus and meningitis, more commonly the syphilitic or serous variety of the latter, and a considerable amount of literature has appeared on pseudotumor. My attention has been called recently to some of the rarer lesions of the brain which may simulate tumor closely, and because of their infrequency may be more likely to cause a mistaken diagnosis, and even lead to operation. Of these lesions, polioencephalitis, thrombosis of the cerebral vessels, and thrombosis of the venous sinuses are especially noteworthy.

The following is a case illustrative of the difficulties that may be caused in diagnosis by polioencephalitis:

A girl, 14 years of age, was referred to me by Dr. F. H. Macfarland, June 24, 1908. She had been well until about six weeks previously, at which time the right lower limb began to grow weak, and in about one week the right upper limb also became weak. The paresis of these two limbs became gradually greater and was associated with nausea, vomiting, headache and unilateral convulsions.

At my examination after her entrance to the hospital the child was weak in the lower part of the right side of the face, and in the right upper and lower limbs, especially in the lower limb. No form of aphasia and no disturbance of sensation were present. The tendon reflexes were exaggerated, the right more than the left, and patellar clonus was obtained on the right side. Babinski's sign was pronounced on the right side.

One of the convulsive attacks was carefully observed. It began with numbness in the right upper limb and soon extended to the right lower limb; the right side of the face was drawn up and held rigidly distorted. The left side of the body was not implicated. The right side of the body was rigid about four minutes, without any clonic movements, and the child was unable to move the right upper and lower limbs about two hours following the attack. She did not lose consciousness.

It is very evident from the record here presented that we had to deal with a focal lesion of the brain. The duration of symptoms for about six weeks; headache, nausea, vomiting, progressively developing right hemiplegia, right-sided convulsions, made the diagnosis of location in the motor region positive, especially as all sensory disturbances and hemianopsia were absent. The symptoms were those of some lesion gradually increasing in size, and more like those of tumor than any other lesion. This diagnosis was rendered more probable by the report from Dr. de Schweinitz, which was as follows:

> Vision 5/6 in each eye. Beginning papillitis in each eye: nasal, upper and lower margins obscured, temporal still visible, veins full. Pupils react normally, no muscle palsies.

Believing that the symptoms warranted an exploration, I requested Dr. Frazier to operate over the left motor area, and the brain was found distinctly diseased in this part. A portion of the tissue was removed, as at the operation it was impossible to exclude glioma, as the diseased tissue, especially after the brain had been exposed a short time resembled glioma. When the flap was first turned back the lesion impressed us as probably one of encephalitis. The microscopic examination revealed polioencephalitis.

The extreme difficulty that may occur in diagnosing between polioencephalitis and brain tumor is well shown by this case, as papillitis is not a common sign of the former condition, although known to occur. I am quite sure that fever was not present at any time while the child was under my observation, and no statement regarding the existence of fever was made by those who had had charge of her in the onset of the symptoms. The temperature charts taken at the hospital were mislaid.

For a time hemiplegia was increased after the operation, but power was regained later on the right side to the extent that the gait showed only a little limp, but the paralysis of the right upper limb was pronounced.

In the report of the committee appointed by the New York Neurological Society to study the epidemic of poliomyelitis occurring in New York during the summer of 1907, embracing an examination of 2,000 cases, Dr. B. Sachs[15] remarked that few cases lent support to Strümpell's view that there is a polioencephalitis closely allied to poliomyelitis; and yet non-purulent polioencephalitis unquestionably occurs, as shown by the case referred to. I have specimens also from three or four other cases.

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[15] Sachs: Jour. Nerv. and Ment. Dis., Oct. 19, 1909.

In another case the diagnosis had to be made between cerebral thrombosis and brain tumor. This usually causes little perplexity, but the following case in the service of Dr. John H. Musser shows that the diagnosis may not be easy. The man had had the symptoms about a year, namely, frontal headache, later left-sided headache, word-blindness, occasional loss of memory, vertigo, nausea, vomiting, subjective weakness of the right upper limb, later paraphasia, some word-deafness, right lateral homonymous hemiamblyopia, dissociation of color sense, and ataxia of the right upper limb. The case has been referred to in my recent paper[16] read at the meeting of the American Medical Association in June, 1909.

The diagnosis between tumor and softening from thrombosis was not easy. The absence of papilledema did not exclude tumor, as I have known glioma of the occipital lobe to reach a considerable size without causing any swelling of the disc. There was unquestionably a gradual increase in symptoms, and this was very suggestive of tumor, but the increase seemed to occur rather abruptly, within a few days, and then no further increase was observed for weeks. This might occur in tumor, and is sometimes seen in thrombosis when the thrombus extends further in the vessel first occluded, or extends into a branch of this vessel. The age of the patient could not help us, for while thrombosis is not uncommon at the age of 60, glioma also may develop at this period.

There could be no doubt as to the position of the lesion. The word-blindness, word-deafness, right lateral homonymous hemiamblyopia, etc., made the first temporal convolution of the left side the seat of the lesion.

The visual disturbance in this case was very interesting. At no time could I find complete hemianopsia. The man could always recognize the movements of objects in the right fields, but he could not distinguish well between objects in these fields, and was positive that he saw them less distinctly than he did in the left fields. Here probably was an important sign in favor of thrombosis. It might readily be overlooked and called hemianopsia, but in my examinations it was never more than hemiamblyopia. It indicated in connection with the other symptoms that a lesion was present which had a considerable superficial extent in order to produce word-blindness, word-deafness, etc., and yet did not extend far toward the optic radiations, which are not remote from the first temporal convolution. Such a lesion would more likely be thrombosis than tumor. Therefore, in the indications that the growth was large but chiefly superficial, and in the rather abrupt increase of symptoms, we had two signs in favor of thrombosis rather than tumor.

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[16] Spiller, W. G.: Brain Tumor, The Journal A. M. A., Dec. 18, 1909, liii, 2078.

After careful consideration, even though the diagnosis of thrombosis of the posterior branch of the middle cerebral artery was made as probable, exploration seemed advisable, as it was at least possible that a tumor would be found. The patient and his relatives wanted some active intervention. I therefore requested Dr. Frazier to expose the brain with the posterior part of the left first temporal convolution as the center of the opening. The operation revealed unmistakable thrombosis in the region diagnosed as affected, and a piece of the diseased tissue excised and examined microscopically showed softening. The man made an excellent recovery.

Another case resembling the one just reported was one I had the opportunity of studying in 1900 in association with Dr. Mills, under whose care the patient was. Dr. Mills has published a report of this case.[17] The man had impaired recognition of objects in the right hand with the eyes closed, diminution in pain and temperature sensations on the right side of the body, word-deafness, word-blindness, paraphasia and right lateral hemianopsia. The first symptoms appeared in the early spring of 1898. The patient suddenly became dizzy and was numb in the right upper and lower limbs. The numbness soon disappeared, but recurred at intervals lasting a few moments at a time. The patient had no other symptoms of importance until he began to have headache in February, 1900. The headache was not continuous, but the attacks of numbness became more and more frequent. Dr. Mills first examined the man on June 2, 1900, at which time he had the symptoms above enumerated. Dr. de Schweinitz found high myopic astigmatism, but no positive optic neuritis, although he stated that the condition was such as to indicate the possibility of an incipient nerve inflammation. The congestion of the optic discs present at previous examinations could probably be attributed to disorders of refraction, and was more marked on the left side. The left side was the side of the lesion.

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[17] Mills: Philadelphia Med. Jour., April 20, 1901.

About June 23, 1900, a decided paresis of the right half of the body developed and operation was decided on and was performed by Dr. W. W. Keen, assisted by Dr. William J. Taylor. An area of softening in the distribution of the Sylvian artery was found and was caused by thrombosis.

The whole condition was evidently of vascular origin. The early symptoms were dependent on impaired nutrition of the parietal lobe, the symptoms which developed acutely about three months before the operation were caused by thrombosis of the branches of the Sylvian artery supplying the first temporal convolution and parietal lobe, and the weakness of the right arm which came on suddenly was the result either of thrombosis of the branch supplying the arm center or of hemorrhage occurring within the softened brain tissue.

The most interesting features of this case were the successive abrupt increases in symptoms, indicating that additional areas of the brain had become affected. The weakness of the right upper limb developed suddenly, not very gradually as is suggestive of tumor, and yet in tumor paralysis may develop rapidly.

Thrombosis of a venous sinus also may cause difficulty in diagnosis, as shown by the following case referred to me by Dr. William Zentmayer. The patient had presented the general symptoms of brain tumor about a year, some of the symptoms preceding the others several months. They were headache, nausea, vomiting and choked discs. As symptoms of localizing value were tinnitus in the left ear, weakness of the right external rectus muscle, some diminution of each patellar tendon reflex and of each Achilles tendon reflex, tendency to go to the right in walking, and, most important of all, diminished sensation to touch in the right scleral conjunctiva and cornea, and to touch and pain in the right forehead, and possibly slight diminution to pain sensation in the right infra-orbital region, without disturbance of the motor supply of the right fifth nerve.

The case seemed to be one of brain tumor, but the localization was not easy. The tendency to go to the right and the diminution of the patellar reflexes were slight and not of sufficient importance to weigh much in diagnosis. The tinnitus of the left ear probably was from ear disease. The only important localizing signs were weakness of the right external rectus muscle and partial impairment of objective sensation in a portion of the right fifth distribution, distinct only in the right forehead and the right eyeball, and more so in the latter. Paresis of an external rectus muscle first observed late in a case is of doubtful diagnostic value. The objective disturbance of sensation in the distribution of the right fifth nerve was, therefore, the most important sign, but this was not complete, varied in different examinations, was only in a small portion of the fifth nerve distribution, and was without any implication of the motor fifth. I could not doubt that the fifth nerve was implicated in the lesion, but the symptoms were not sufficient to justify exploration of the base of the brain, especially as the patient had never had signs of irritation of this nerve, namely, pain or tenderness to pressure over the points of exit, and spontaneous pain. A glioma of the pons could cause the slight disorder of the fifth nerve without other symptoms if the tumor were small. I have recently called attention to this possibility.

The study of this case did not seem to warrant a basal operation, and, therefore, in the hope of relieving the patient of headache, nausea, vomiting and vertigo, I requested Dr. Frazier to do a subtemporal decompression, believing that later a basal operation would be called for if the symptoms became more decided.

The hernia of the brain that developed caused much pressure of the right frontal lobe and motor region by the protrusion of the brain through the opening, and the cutting off in this way of the blood-supply. Softening of the brain in the area of the occluded vessels occurred, and a purulent meningitis was produced by the patient handling the dressings and the wound before he could be restrained.

The findings in this case were extremely interesting. The necropsy was performed by Dr. H. T. Karsner, who kindly gave me the brain for examination.

A thrombus was found in the right inferior petrosal sinus extending into the cavernous sinus, and causing a depression in the pons. It was about an inch in thickness and about an inch and a quarter in length. The clot was shown by the microscope to be well organized. Whether the thrombus had its origin in some disease of the sinuses communicating with the nose or not, it is impossible to say.

1906 Chestnut Street.

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OPERATIONS FOR BRAIN TUMORS[1]

By WILLIAM G. SPILLER, M. D., Philadelphia, Pennsylvania

In considering operation for brain tumor we should bear in mind that we are dealing with a lesion that must in course of time prove fatal, and usually within a short time. Tumors of the pituitary body frequently seem to be of slower development than are new growths in many other parts of the brain. Acromegaly is a condition of slow development and long duration, and while a tumor of the pituitary body is not found in every case of this disease, it not rarely seems to be the cause, and when present does not appear to hasten greatly the fatal termination. Frequently cessation of menstruation is the first sign of tumor in this region of the brain, and it may precede the other symptoms for many years. A tumor of the pituitary body may attain considerable size without causing many symptoms, and a bitemporal hæmianopsia may be its only sign for a long time. The sella turcica is enlarged by pressure from the growth, and the hæmianopsia may in this way become less by relief of pressure on the chiasm. This variation in the hæmianopsia sometimes perplexes the clinician. The third ventricle is above the pituitary body, and a tumor may grow to large size in this ventricle before it produces many symptoms. For these reasons one may hesitate before attempting to remove a tumor from this region. The operation in whatever way it may be undertaken is very serious, and while a few successful cases are on record, we can not forget the grave danger in surgical intervention. It is important to keep in mind that because of the relations referred to above, grave symptoms of tumor of the pituitary body are likely to indicate a large growth, and that operation would be much more promising if bitemporal hæmianopsia were the only sign. Bitemporal hæmianopsia may be produced by meningitis, fracture of the base of the skull, or aneurysm, but is strongly suggestive of tumor when developing slowly and without a previous trauma of the head, especially is this true if to the bitemporal hæmianopsia complete blindness of one eye be added, indicating complete loss of function of the fibers in one optic tract, or if primary optic atrophy instead of papillœdema be present. In some instances, as in a case reported recently by Gowers, primary optic atrophy may be observed on the side on which the tumor is larger, and papillœdema on the other side where the tumor is sufficiently far from the papilla to permit papillœdema to develop. In this case skilfully diagnosed by Gowers, operation was performed by so experienced a cerebral surgeon as Horsley, and yet it resulted fatally.

Statistics as now given regarding operation in cases of brain tumor are unreliable, because infiltrating and well defined tumors are included in the same tables, and little attention is paid to the mortality depending upon the position of the tumor.

The statement that operation is more likely to be successful when the tumor is small hardly admits of dispute, but it is not always easy to make the diagnosis in the early stages, and frequently the patient is not seen by the neurologist at this period. Symptoms which to an experienced physician are very suggestive of tumor, may be attributed to gastric or hepatic disorder, to nephritis of pregnancy, or some other cause; and tumor especially of the cerebellum, may produce a condition of the retina resembling albuminuric retinitis.

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[1] Remarks made with a lantern demonstration at the annual meeting of the Medical Society of the County of Rensselaer, Troy, Dec. 15, 1900; and later at a meeting of the College of Physicians of Philadelphia.

It may not always be remembered that sudden death is common in cases of tumor in any part of the brain, but chiefly when the tumor is in the posterior cranial fossa. There is less room here than in any other part of the cranium for a new growth, and increase of intracranial pressure therefore occurs more rapidly, and the respiration and cardiac action may suddenly become seriously impaired.

It is wise always to present the condition clearly to those who may be responsible for the patient, and often to explain the condition to the patient himself if he be intelligent. One will frequently find that an intelligent person will demand that the chance of operation shall be given to him, even though he fully realize how serious a surgical intervention may be. He recognizes on the one hand that he has a cerebral lesion which must prove fatal, and that the end may come suddenly within a few days, a few weeks, or after a longer period; that while he lives he must suffer great impairment of function in important structures and probably great discomfort, especially in headache, nausea, and vomiting. On the other hand he knows that surgery may give him a chance for his life or for temporary relief, and he gives no choice to the physician, but demands as his right that the chance be given to him. More than once the surgeon will be told by the patient that no effort to remove the tumor is to be slighted, and that death would be preferable to a continued existence in his deplorable state.

There is no denying that the mortality in operations for brain tumors is high, but it is not so high as the mortality of tumor cases without operation, which approaches 100 per cent. Surgical technique has been so greatly improved in recent years that operations for tumors have become almost a special branch of surgery. The success of surgical intervention will depend chiefly on the correctness of the diagnosis, on the position and size of the tumor, and on the skill and experience of the surgeon. The neurologist must strive to make his diagnosis at an earlier period in the development of the tumor, and should not wait until all the important signs of increased intracranial pressure have developed, and to these are added many sharply focalizing symptoms. In the early stage of tumor general signs of intracranial pressure may be entirely absent, and there may be no headache, no nausea or vomiting, no papillœdema. A slowly developing focal symptom or focal group of symptoms may be sufficient for exploration, and this is especially true if the lesion be diagnosed as being in an accessible portion of the brain. Convulsions confined to one limb, or both limbs on one side of the body, or also to the face on the same side, and occurring repeatedly, indicate irritation of the opposite motor region of the cortex; and while the source of this irritation is not always detectable, a cause must be present. Such convulsions are not always produced by tumor, but frequently are so produced, and at least twice in the writer's experience were the only important sign of a tumor discovered at operation. The motor region is easily exposed and little damage is likely when an osteoplastic flap is made, provided the patient's general condition permits operation. The scar from this operation may almost disappear and may be detected only by careful search, even a short time after the operation. It is probably a wise procedure to expose the motor cortex when the convulsions are strictly Jacksonian in type and of frequent occurrence. Unquestionably a tumor will not always be found, but if we expect to operate early we must be prepared to take more chances than in a case easily diagnosed by a first year medical student, and yet rashness is not justifiable.

A paræsthesia confined to the distribution of the fifth nerve, followed after several months by anæsthesia in the distribution of this nerve and paralysis of the muscles of mastication on the same side, indicates a lesion paralyzing the fifth nerve. In the writer's experience this symptom-complex has been produced by basal syphilitic meningitis, but it is more likely to be caused by tumor. When the Wassermann reaction is negative and the anti-syphilitic treatment has failed, the diagnosis of a tumor in the region of the pons may be considered probable, but a negative Wassermann test does not necessarily exclude syphilis of the brain. It is not easy to determine at first whether the tumor be in the pons or growing from the dura over the petrous portion of the temporal bone. Glioma within the pons does not destroy all the area in which it develops, and trigeminal disturbance may be the most pronounced sign of glioma of the pons, but some weakness of the opposite limbs is likely to develop soon. Tumor growing in the region of the petrous portion of the temporal bone usually causes pain in the distribution of the trigeminal nerve from irritation of this nerve, but in one of the author's recent cases pain was absent. A tumor in the middle cranial fossa may implicate the bone, and in the writer's experience has extended into and partly filled the nasal cavities.

The greatest difference in symptoms may be occasioned by a tumor just above the tentorium and one just below this membrane. When below, the symptoms usually are very characteristic, and are such as cerebellar ataxia, unilateral nerve deafness with facial palsy on the same side, early papillœdema, nystagmus, vertigo and severe headache. A tumor growing above the tentorium, from the floor of the middle cranial fossa, may develop to a large size with few symptoms, if it does not implicate the trigeminal and abducent nerves. Here it forms a nest for itself in the basal part of the occipital lobe by atrophy of the overlying lobe, and as the optic radiations do not extend to the base of this lobe hæmianopsia may not be present. Papillœdema and headache may be the first signs of a growth in this region, and when the patient comes under observation the former may have attained so great a degree as to mask a hæmianopsia that had been present. The tumors at the base of the occipital lobe are very likely to escape detection when small.

The endothelioma is probably the most favorable tumor for operation. It does not infiltrate the brain, it grows from the dura and is well defined, and is less likely to recur than is almost any other form of tumor. It seems a strange perversity of nature that most of the tumors in the cerebral hemispheres, where tumor is most accessible, are gliomata; and that most of the basal tumors, and therefore in regions of greater danger, are endotheliomata. Mallory regards the tumors commonly considered as fibromata or fibrosarcomata, as endotheliomata. These tumors occasionally develop over the cerebral hemispheres, and the most successful surgical cases are those in which a growth of this character is found. Endothelioma may infiltrate the overlying bone, and the hæmorrhage then from the bone is severe when the bone is cut. A distinction should be made between a slowly growing and a rapidly growing endothelioma.

In the later stages of tumor the pressure at the base of the brain may cause partial obstruction of the ventricular cavities, and hydrocephalus. The symptoms then become so indistinct and indicative of a widespread lesion that a focal diagnosis may be impossible. The tumor often is so large at a late stage when operation is attempted that it can not be removed, or if removed the patient dies within a few hours after the operation, if not on the operating table. The pathologist too often finds a tumor that should have been removed and had remained undiagnosed, and on the other hand the surgeon sometimes removes an enormous tumor which from its character and situation should have given a favorable prognosis if permission for operation had been granted earlier.

It is not easy to explain certain signs of brain tumor. Failure of light reflex and bilateral exopthalmus have occurred in the writer's experience in tumor of the temporal lobe. Again we may see improvement in the condition of a patient who has a large tumor of the brain, especially if iodid has been given. Why this occurs we do not know. The tumor has not diminished in size, indeed it may have increased in size, and yet intelligence may take the place of stupor, and hopes are raised that soon perish. Seldom do periodic exacerbations occur in such an extraordinary manner as in the recent case reported by Rudolf and Mackenzie in which they seemed to be related to the menstrual period.

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STUDIES IN NEUROFIBRILS.

By S. D. W. Ludlum, M.D.,
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The studies in this paper are confined to normal and pathological material; there has been no attempt made to investigate the development or regeneration of neurofibrils. After the publication of the Cajal-silver impregnation for the fibrillar structures in nerve cells there was at once a stimulation displayed in the histological studies of these structures. Marinesco very shortly published a paper on the arrangement of fibrils in the cell and upon the effect of cutting the axone on their continuity, also, he wrote upon the effect of ligation of the abdominal aorta upon cells in the cord. Both of which showed degeneration.

In the same year he showed changes due to postmortem conditions. The next year he studied the root ganglia and wrote on the evidence of regeneration of fibrils in cells.

These papers and others showed nothing which was epoch-making in cellular pathology, but confirmed much that might have been predicted from Nissl and other preparations, but showed with more accuracy the changes found in various nervous conditions. In 1905, Professor Charles Schaffer wrote still farther on the subject. He was of the opinion that fibrils anastomose in the processes and that main fibrillar trunks from processes come into a cell and break up into divisions and anastomose, and that Nissl bodies occupy the space between them. He described an outer parallel arranged layer and an inner or network appearance. Much work was done by persons working in various laboratories, and among them I published a paper on the relationship of neurofibrillar changes to mental disease. It is an elaboration of that idea and the connection which other diseases assume toward this particular nerve-cell histology, which I now wish to describe.

Shortly after the Cajal-silver stain became established Donnagio published his method of fixing in acetone and staining with thionin and afterward Bielschowsky brought out his silver impregnation used upon frozen sections.

I have made experiments with the three methods each on rabbit, dog, and human material. The Cajal method necessitated using small blocks of tissue, but the impregnation was not uniform from periphery to centre of the block. The Donnagio method would stain beautifully one portion of a section and show nothing in the other parts. With this method it was difficult to make enough good sections to develop any very definite statements.

Of the three methods the Bielschowsky was by far the most reliable. In this, sections were cut from frozen blocks, then impregnated, and in this way greater uniformity was obtained, and results shown which could be depended upon. The three methods when all were successfully performed on one kind of tissue showed identical results, and therefore I have used the Bielschowsky method as an index of the changes taking place because of its greater efficiency.

The method is not simple, but can be successfully carried out if accurately done. The method with some changes in the time and washings is as follows:

Tissue to be used must be fresh or of formalin hardening of not more than forty days' fixation in the formalin. Sections are cut as thin as possible on a freezing microtome, placed in water. Immersed in 2 per cent. $AgNo_3$ in the dark for twenty-four to thirty-six hours. Then transferred to a solution of $AgNo_3$ × (NaOH, two drops) precipitate; this solution cleared by adding 10 per cent. AMOH solution; taking care to have just enough ammonia and none in excess. This is done by adding a drop at a time until precipitate is dissolved. Sections in solution for seven minutes. Wash in water for one second. Formalin, 20 per cent. for ten minutes or until color becomes black and then begins to lighten a little. Wash in water five minutes; color lighter to brown.

Gold chloride, 1 per cent., 5 c.c., to which add 2 drops of acetic acid. Sections tone to silver gray; remove as soon as toned. Wash one second in water. Sodium hyposulphite, one second, tone lightly silver, care not to keep in too long else it becomes darker. Wash one second in water. Alcohol, 95 per cent. Alcohol, absolute. Xylol. Mount in balsam.

Sections when properly prepared will be a silver gray. The extracellular and intracellular fibrils being the only thing taking stain except the bloodvessels. Although it has sometimes looked as though some of the extracellular fibers might be neuroglia. The nucleus shows as a clear round space in which can be seen the nucleolus with usually four or five distinct granules contained in it. The processes of the cell can be traced some distance, but never to the point of contact with another cell, that is, in the human adult system. Bethe has shown the connection in the bloodvessels and corpuscles show as dark colored structures and can be easily differentiated.

Betz cells and the larger cells in the human cortex appear with a parallel arrangement of fibrils throughout the length of the cell; a cross-section of the cell would seem to show the cut ends of these structures. On the other hand, cells in the medulla and thalami display a reticulated arrangement about the central part of the cell and a parallel arrangement in the periphery, branching and anastomosing as they go into the processes. In the processes the general arrangement is parallel but now and then one gets a picture indicating that one fibril will connect with another fibril in the process anastomosing, or it may be simply crossing or twisting. In the cord the cells are reticulated in the central part and parallel in the periphery, but not markedly so as in the medulla. The cord when cut longitudinally instead of transversely shows that the cells are more or less equilateral in shape and that the fibrillar arrangement is quite similar looked at from any plane. And taking for example a particular instance of a case of paresis, one form of cellular degeneration is well shown. This case was carefully stained by means of the Cajal stain, Donnagio stain, and the Bielschowsky method. In each similar results were obtained. There was yellow pigment in the cells in all parts of the nervous system, more in the thalami than in the cortex. The pigment forms in the meshes of the fibrillar reticulum. In early deposition there is found at first a single drop in a cell, in those farther advanced more drops, and so on a greater accumulation until a large part of the cell may be taken up by the pigment. As it accumulates, the droplets seem to increase in size and press one against the other. In doing this the fibrils would appear to be impinged upon and other sections would indicate that this pressure disintegrated the fibril and a granular degeneration resulted, quite analogous to the after effects of cutting a peripheral nerve causing the degeneration of the axone.

This granular degeneration then spreads to all the reticulated portions of the cell about the nucleus, hence to the periphery, but stopping at the processes, and the eventual picture is a cell entirely granular with uninvolved fibrils in the cell processes except in very advanced stages, when these too were changed. The nucleus may be pushed to one side and the nucleolus contain a number of small granules; these granules are never seen numbering more than four in a normal cell, but exceed that in pathological states. The small cells in the cortex appear to break up in this way before the large cells. In these degenerated cells the granules take the stain in the same manner as fibrils become impregnated.

In the cortex side by side will be found a normal cell and an abnormal or degenerated one. In the degenerated cell, fragments of broken fibrils will remain and the arrangement in the cell will show that it is not artefact, but there are many cells which might appear to be degenerated but are really poorly stained, hence it takes a good deal of experience with these cells in the present state of the coloring to give an accurate opinion as to the change taking place. In the portion of the cell where the reticulated parts are about the centre, it would seem that the mesh is not made up of connecting fibrils but rather of the crossing of fibrils by others giving the appearance of a mesh similar to wire screening.

In sections from the thalamus in which cells are a good size some fibrils are observed thicker than others in apparently good cells in a pathological case. These thicker fibrils advance into the processes of the cell; they there break up into smaller ones and some go into divisions of the processes. As a general statement the fibrils in the thalamic cells are finer and interlace more than in cortical cells.

In all the stains the pigment takes a slight yellow color, as in the Nissl stain, and I would say that Nissl preparations of all these specimens showed pictures which coincide with the fibril stains, one being a negative to the other's positive. It has long been recognized in the Nissl specimens that in degenerated conditions the change in the Nissl bodies is in the cell body and does not extend into the processes. In this case of paresis most all the cells in the medulla seem affected, more so generally than in the thalamus. In the lower processes of these cells fibrils can be traced for long distances, but they do not give off anastomotic branches, but do cross one another and would give the appearance of anastomosis; the only branching is in the region of divisions of the processes. A few cells are seen in which the entire centre of the cell has disintegrated and appearing to be absorbed, leaving only the cortex of the cell with a few fibrils in the periphery. These may be the shadow cells sometimes described.

This is a type of cellular degeneration judging from the fibrillar condition which shows a marked degree of degeneration; it is similar in type to that which takes place in similar conditions, only very much more marked, but the kind is the same. It is that of a long-continued katabolism. This is seen in several cases of cerebral hemorrhages of old age arteriosclerosis, paralysis agitans, which I have examined. It makes one think that paresis is really only another form of old age, congenital nervous weakness, perhaps. There is with advancing years a deposition of yellow pigment in the fibrillar meshes; this appears to cause pressure atrophy of fibrils as it increases in amount, consequent to this is a granular degeneration of the structure, for in three cases examined of arteriosclerosis it was found that in cells where there was little accumulation of yellow pigment there was also little degeneration; but where there was much yellow pigment in a cell there was much granular degeneration of fibrillar structures. In one case of paralysis agitans a similar picture was observed, except that scarcely a cell escaped the load of yellow pigment. In the oncoming of old age, where it is gradual and natural and not augmented by disease or strain, the development of pigment is so gradual that very little alteration takes place in the integrity of fibrils; they are pushed aside into bundles of strands. The process seems to be comparative to the condition of the mental faculties, the one being the index of the other, judging from the histories and the pathological findings.

Now considering other conditions directly and indirectly affecting nerve cells, conditions which are more or less acute in type, such as typhus fever, purulent meningitis, carcinoma, syphilis, and tuberculosis of the brain. I find in these cases a granular breaking up, but there is no yellow pigment except the case be of advanced years, and the degeneration is different in type in that the fibrils display a fine granular breaking up and not the coarse disintegration described in the cases mentioned where we had the factor of chronicity introduced. The process is one of an acute change apparently; all these cases displayed the appearance, the scarcity of yellow pigment, and the finely granular condition of fibrils. The nucleus is apt to be swollen and displaced, some cells show a diffuse, finely granular condition of fibrils throughout the cell, others with involvement only of the base of cell; some cells in this acute affection show a swelling of the fibrils which probably precedes a breaking up.

In any of these acute cases where there was found any yellow pigment, and the case was too young for the normal appearance of pigment, there was the history of a previous strenuous existence.

In the cord the anatomy of the intracellular structure is similar to the medulla, except that the reticulation is not so marked, fibrils are more parallel in arrangement, and seem to go in one process, cross over the cell, and go out another.

In a case of syringomyelia, in which I examined the cortex, there is a suggestion that the cells are lessened in number; but those cells present, while they show an unusual amount of pigment, yet may be only physiological, and according to age for the fibrils are quite normal, there being little breaking up of them, and when the pigment is present they have been pushed aside, as is seen in senile or age advancing conditions.

In tabes (two cases) the cord shows some degeneration in cells, not any particular group, although the column of Clarke is always affected. Longitudinal sections of the cord show that the cells present the same contour and size as when seen transversely, and in addition the axis cylinders are plainly seen degenerated in the column of Gowers and dorsal column. The slightest change in the axis cylinders is seen with this stain; changes which do not show with Weigert staining of myelin sheaths.

A few cortical cells are broken up in tabes but not more so in the paracentral or parietal lobules than in any other portion of the cortex. This is true in three cases examined. Someone has claimed that secondary changes in the paracentral region are seen, due to sensory degeneration acting upon the motor cells. Parenthetically, it must not be too strongly urged in such work to use as fresh material as possible, for long fixation in formalin does alter the intracellular structure.

In a case of pernicious anemia the cells in the cortex did not appear to be damaged to any appreciable extent, but cells in the cord in all cell groups are affected. There was very little yellow pigment in the cortical cells but a good deal in the cord cells. The appearance was one of acute change regarded from the standpoint of fibrillar degeneration, as the granulation was of the fine type. Cells in the cord of old people show a similar accumulation of pigment as in the cortex.

An interesting fact in relation to the use of this stain is in the detection of perhaps reflex influence from the periphery. In a case of arthritic atrophy in a man of forty years, the arms were markedly affected but the legs only slightly; and in examination it is found that the upper thoracic and lower cervical is the only part of the cord showing marked cellular changes, and in all cell groups. This would seem to indicate that the arthritic condition of the arms reflexly affected the cells in the region of the brachial plexus. The cells were natural in the processes, but the interiors were quite bare of detritus and would give the suggestion of a slowly progressing atrophy either from disuse or reflex influence. These cells were carefully studied transversely and longitudinally; those in the lumbar region were about the same average size in both dimensions; those in the thoracic are about the same size transversely, but longer in longitudinal views.

In eight cases of insanity we have the following etiological factors: Uremia, carcinoma, syphilis, exhaustion, arteriosclerosis. In all these cases the process has apparently been of some duration, judging alone from the microscopic findings, because the granulation is coarse, not the fine dotted appearance of the acute condition, and the histories of the cases would bear this out.

Scarcely a case can die without some mental change at the end, no matter what the disease. It would not be justifiable to assume that mental activity is an influence derived from neurofibrillar filaments and appreciated in our consciousness, but it can be stated that by studying fibrillar conditions in cortical cells a fairly accurate idea can be arrived at of the mental state at the time of death.

It would seem that there are two forms of fibrillary degeneration—one the coarse breaking up into large granules, and the other the fine granulations. The one occurring coincident with deposition of yellow pigment, or in the prolonged course of a chronic disease; the other as a result of an acute febrile attack or other quickly terminating disease.

The reparative power of fibrils in convalescence is unknown as yet. I would say that neurofibrils are affected in all cases where death occurs from a toxic or wasting disease. I have no doubt that there are fibrillar changes in the course of severe diseases which recover, and that there is a degeneration and regeneration in the fibrillar structures, especially where there is delirium and mental phenomenon, but that is necessarily hypothetical as yet. Any disease which is severe enough to cause death will show fibrillar changes in some cells at postmortem. Many cases of insanity show fibrillar changes throughout the nervous system. These were all cases with a clear etiological factor. In conditions of paranoia I doubt if in life we would find fibrillar changes to cause the mental obliquity. Or in obsessions and such mental deviation would we find fibrillar changes, but in toxic insanities, deliriums, manic states and depressed states, paresis, any mental confusion, we do find fibrillar changes, and in these conditions many authors claim the etiological factor to be a toxemia or a faulty glandular secretion, etc., which is sufficient to cause fibrillar changes.

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SPECIAL FEATURES CONCERNING MULTIPLE NEURITIS.*

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As anything like an historical retrospect concerning the subject of polyneuritis or multiple neuritis, is hardly germane to the special considerations at issue in this paper, I shall limit myself to an encomium on the master mind who in 1879 first untangled the chaotic reasoning on the etiology of Kinderlähmung—von Leyden.

That the knowledge on the subject prior to this date was certainly "without form, and void" is amply evidenced by von Leyden's statement:[1] "Merkwürdiger Weise ist aber hieraus niemals ein Bedenken gegen die spinale Natur der Krankheit entnommen oder auch nur die Frage erörtert worden, in wie weit periphere Processe in den Nerven und Muskeln an diesen Erscheinungen betheiligt sein können. Das Dogma der Poliomyelitis wurde nicht angetastet. Weil die Kinderlähmung nachweislich mit einer Rückenmarkserkrankung und einer Atrophie der grossen Ganglienzellen in den Vorderhörnern einherging, weil bei ihr die Muskeln jene Erscheinungen der Entartungsreaction darboten, so musste auch für die anderen ähnlichen Processe, 'Mit Sicherheit' auf die gleiche Läsion im Rückenmark geschlossen werden. Es bildete sich die Anschauung aus, dass gerade die Erkrankung der grossen Ganglienzellen am sichersten und schnellsten jene tropischen und electrischen Störungen in Nerven und Muskeln hervorrufen, welche an den schweren traumatischen und rheumatischen Lähmungen studirt waren: die Frage, ob es nicht anders sein könnte und sich in einigen Fällen anders verhalte, ist kaum mehr erörtert worden."

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* Read before the Section on Neurology of the XVI International Medical Congress at Budapest, August 29 to September 4, 1909.

It is my purpose at present—thirty years after the blazing of this trail—to deal with a few special features of multiple neuritis, to wit:

(*a*) Argyll-Robertson pupil.
(*b*) The involvement of cranial nerves.
(*c*) Involvement of the vagus.
(*d*) Changes in the upper motor neuron system.
(*e*) Involvement of the bladder and rectum.
(*f*) Muscular spasms.
(*g*) Inequality of the pupils.

I report in brief the histories of the following cases. I have only selected from the autopsy records such statements as seem relevant to multiple neuritis, such as malignant disease, tuberculosis, nephritis—parenchymatous or chronic interstitial, etc.

Case 1.--No. 419, A. M., female, white, aged forty-eight years; was admitted to the Philadelphia General Hospital (Dr. Spiller's service) August 13, 1907. Her family history was negative. Her chief complaint was difficulty in walking.

From twenty-four years of age she drank beer in moderation, two glasses every other day. She had had gonorrhea, but there was no history of syphilis, and she had never had a miscarriage.

Her trouble began about 1903, and was at first characterized by numbness in her feet, extending later to her legs. This was followed in about one year by pins and needles sensation. During the year prior to admission she had sharp shooting pains in her lower limbs, together with difficulty in walking and weakness in her knees. She had to be supported in order to walk. She swayed slightly when standing with her eyes closed.

Examination on admission showed normal condition of all cranial nerves.

The upper extremities showed some slight weakness and absence of biceps and triceps tendon reflexes on each side. No pain on pressure was present.

The lower extremities were weak and somewhat wasted, and patellar tendon and Achilles tendon reflexes were absent. Nowhere on the body was there any disturbance of sensation.

She improved in strength and general condition, and was discharged at her own request November 19, 1907. The diagnosis at this time was thought to be most probably tabes dorsalis.

She was re-admitted on January 4, 1908, at which date it is recorded that her eyeballs were prominent. Convergence was fairly well performed. There was a tendency to fix with the left eye and in convergence to allow the right eye to swing out under cover. The pupils were equal and the irides reacted to accommodation and convergence, but there was no reaction to light.

The left nasolabial fold was more pronounced than the right and the left corner of her mouth could be elevated better than the right. In corrugating her forehead or closing her eyes there was no difference detected between the two sides.

The upper limbs were flabby and without tonicity. She could move them in any direction, the power in the left being fair and slightly greater than that in the right. There was tenderness on pressure over the muscles and nerve trunks. Ataxia of moderate degree was present in each upper limb. There were no contractures and no atrophy. The biceps tendon and triceps tendon reflexes were absent on both sides.

The lower limbs were flabby and atonic as the upper limbs. Voluntary motion was possible in all directions. The slightest pressure over muscles or nerve trunks elicited great tenderness and severe pain, as did also forcible extension of her limbs. Her power was greatly diminished. In the heel to knee test there was no ataxia demonstrable. The patellar tendon and Achilles tendon reflexes were absent. Babinski reflex was negative. Her mentality was fair.

There was no loss of sensation for touch, temperature, or pain, and muscle and stereognostic sense were normal.

On January 9, the day before her death, it is recorded that she had incontinence of bladder and rectum, but her weakness was so extreme at this time, together with the fact that she was suffering from a severe diarrhea, that I hardly think this can be reckoned as due to any disturbance of the nervous mechanism of defecation or micturition.

She died January 10, 1908.

Sections of the spinal cord from about the fourth cervical, seventh cervical, and third lumbar segments were stained by the Weigert hematoxylon, hemalum-acid fuchsin, and thionin phenique methods, and upon examination proved normal.

The median and musculospiral nerves presented a few swollen axis cylinders seen by the hemalum-acid fuchsin method. A nerve from one of the feet showed a swelling of the axis cylinders greater in intensity than that seen in the median or musculospiral nerves. A piece of muscle from one of the feet stained by the hemalum-acid fuchsin method shows a great increase of muscle nuclei and in many muscle fibers a complete loss of cross-striations.

Owing to the Argyll-Robertson symptom, sections were made, both transverse and longitudinal of the optic and oculomotor nerves on both sides. These were stained by the Weigert and hematoxylin-eosin methods. The optic nerves proved normal.

The oculomotor nerves showed many swollen axis cylinders and some nerve fibers in which the axis cylinder could no longer be distinctly seen, and in which the fiber appeared granular.

Serial sections through the oculomotor nucleus demonstrate a group of cells located in the raphé just anterior to the aqueduct of Sylvius, which show axonal degeneration. The large groups to the right and left are mostly in good condition. This central group cannot with certainty be called the oculomotor nucleus, as there are no nerve fibers visible passing in the direction taken by oculomotor fibers.

Case II.—No. 425, T. B., male, white, aged forty-seven years; was admitted to the Philadelphia General Hospital (Dr. Spiller's service) December 13, 1907.

His family history was negative. He admitted the usual diseases of childhood and moderate use of alchohol, but denied venereal disease.

For two months prior to his admission he had had sharp pains in his legs. He has also had difficulty in micturition—the stream being hard to start, and during the passage of urine he has suffered pain referred to the head of his penis. At times he had dull abdominal pain, which occasionally became sharp.

For six or seven months before admission he had been working in a chemical works.

The following observations were noted on his first examination: The right pupil was larger than the left and both irides reacted very sluggishly to light, but well to accommodation and convergence. The other cranial nerves were normal. The gums showed a blue line and were heavily coated with a bluish, black material. The upper limbs showed some weakness of the extensor group, but the action of the supinator longus and the other muscle groups was unimpaired on each side. A moderate amount of incoördination, manifest in the finger to nose test, was present in each upper limb. There was no wrist-drop. The biceps and triceps tendon-jerks are exaggerated on each side. Sensation in the upper limbs was unimpaired.

In the lower extremities voluntary motion was possible in all directions, although decided weakness was present. There was no ataxia in the heel to knee test. The patella and Achilles tendon reflexes were exaggerated on both sides. Patella and ankle clonus were obtained on each side. The Babinski reflex was not present on either side.

On January 21 I recorded on his history: "Pupillary response not obtained to light, though the iris at times seems to dilate paradoxically after the light has been thrown on it for a few seconds."

The patient was transferred to the erysipelas ward from January 1 to January 17, on account of an erysipelatous inflammatory area on his right cheek. From January 25 on he steadily became weaker, and died on January 31, 1908.

Sections of the spinal cord were made at the levels of the seventh and eighth cervical and third lumbar segments and stained by the Weigert hematoxylin, hemalum-acid fuchsin, thionin phenique, and Bielschowski methods.

No abnormality was noted by the Weigert or Hemalum-acid fuchsin methods. In the thionin preparations there is possibly more pigmentation of the anterior cornual cells than might be expected at the patient's age. The Bielschowski stain likewise shows the hyperpigmentation, but also demonstrates a normal condition of neurofibrils in most of the anterior cornual cells and their dendritic processes. The tigroid substance of the anterior cornual cells is well preserved in the lumbar region, but there is axonal degeneration in the cervical enlargement, especially on the right. Unfortunately there were no specimens of peripheral nerves or muscles taken at the autopsy.

Examination of the anterior and posterior nerve roots attached to the cord shows them to be normal, but this is perfectly consonant with a severe grade of distal neuritis, as is seen in Case XVI.

Case III.—No. 122, A. C., male, white, aged sixty-four years; was admitted to the Philadelphia General Hospital (Dr. Mills' service) December 3, 1902.

His mother, father, and three sisters died of tuberculosis. He denied venereal disease, but said he was a constant drinker. For the past five months he had been feeling poorly. Ten weeks before his admission he was taken with severe pains in both upper and lower limbs. A fever developed at this time and for two weeks he was delirious. About four weeks after the onset of pain he began to lose power in his upper and lower limbs. On admission he had absolute quadriplegia.

The irides reacted normally to light, accommodation, and convergence. There was no nystagmus.

The upper limbs showed much atrophy of the muscles of the arm, forearm, and hand, the left being more wasted than the right. The joints of the fingers were painful and the right wrist was somewhat swollen. The fingers were cyanosed and showed at several of the terminal phalanges brownish discoloration. The tendon reflexes of the upper limbs were lost and there was decided tenderness in the muscles on pressure. The tactile, pain, and temperature senses were all preserved and normal.

The lower limbs were not so much wasted as the upper. The muscles were tender on pressure, and the slightest movement of the lower limbs caused excruciating pain. The various forms of sensation seemed preserved, with the possible exception of loss of temperature sense in the feet. There was loss of tendon reflexes and no Babinski phenomenon on either side.

On December 17 it was recorded that the irides no longer reacted to light.

On December 27 it was noted that the skin of the hands, especially the dorsal aspect, was glossy and thin; also he had no control over his bowels, and at times had incontinence of urine.

He died on January 3, 1903.

Sections from the paracentral lobules stained by the thionin phenique method showed that the large ganglion cells were in normal condition. Occasionally there would be found a cell in which the tigroid substance was badly altered, but this was in all cases noted a degeneration of a pigmentary kind and entirely consonant with the patient's age.

Sections from the lumbar and cervical enlargements, stained by the Weigert hematoxylin, hemalum-acid fuchsin, and thionin phenique methods, showed a normal condition of the white matter, but considerable axonal reaction in the anterior cornual cells.

Cross-sections were made from the right and left sixth nerves, the right seventh, both optic and both oculomotor nerves, and stained by the hematoxylin-eosin method.

The optic nerves appear normal. Occasionally there is seen an axis cylinder much larger than the neighboring axis cylinders, but the difference is not sufficient to be called abnormal in view of the fact that there are a moderate number of axis cylinders in the optic nerve which normally are large by comparison with the usually minute size found there. There are some swollen axis cylinders in the sixth nerves and in the right seventh, although no clinical evidence existed of neuritis in these distributions.

The oculomotor nerves of both sides contain many moderately swollen axis cylinders, and in comparison with normal oculomotor nerves show a decided difference.

Cross-sections of the right median nerve were stained by the Weigert hematoxylin and the hemalum-acid fuchsin methods and teased Marchi preparations were also made of the right median nerve. In all these nerves pronounced changes were demonstrated.

Case IV.—No. 257, A. C., female, white, aged thirty-two years; was admitted to the Philadelphia General Hospital (Dr. Mills service) October 16, 1904, and died November 20, 1904. Her father died two years before her admission, of carcinoma of the stomach; two sisters died of puerperal sepsis. Her husband denied all venereal disease.

For more than ten years the patient had been a hard drinker of both beer and whiskey, getting drunk about twice a month. Four years before her admission to the hospital she was delivered of a still-born child. In the next two years she had two living children, each dying under one year of age. Seven weeks before admission she was delivered of a seven months' child, which was also still-born. Prior to the onset of labor she was troubled with vomiting and was drinking her usual amount of alcoholics, which was said to be about eight glasses of beer and a half-pint of whiskey daily.

She was in bed for about two days, and was then around for two days, when she was compelled to go back to bed because of swelling and pain in both feet and legs. Loss of power in her hands and arms and swelling of her face soon followed. Her legs were painful below the knees, especially on their inner aspects. Her bowels were regular and her appetite remained fair.

About three weeks after the swelling of the feet was first noticed she began to ask foolish questions and make irrational remarks, such as inquiring whether her father, who had been dead for two years, was still in the house, and whether he had gone to work; she also asked that her sister, who was also dead, should be sent upstairs to see her.

Examination showed the patient to be a well-developed woman, with slightly flushed face, apathetic expression, and semiclosed eyelids. She turned her head occasionally from side to side, but was otherwise perfectly quiet, lying with her feet turned out, legs straight, fingers slightly flexed upon the palms, and hands upon the forearms. She muttered occasionally about members of her family and made irrelevant remarks. She answered questions with fair intelligence, however, and did as she was told; she lapsed at times into sleep. Her gaze was generally fixed; her pupils were small and equal, but reacted sluggishly, if at all, to light, and but slightly to accommodation. Ocular movements were good in all directions.

The tongue was protruded evenly without difficulty and was not tremulous, but presented a considerable white coating and a stringy mucus.

Her right forearm could be feebly flexed against resistance and her left was even weaker. The grip of the right hand was poor; that of the left was still less. Neither hand could be extended on the forearm. Sensation to pain and touch in the upper extremities appeared to be preserved. Pressure over the bellies of the muscles of the arms caused pain, as did also rapid passive movements. The reflexes were abolished.

The legs could be slightly flexed, and the feet slightly rotated. Otherwise the woman was powerless to move herself in bed. Manipulation of the feet, as in efforts to elicit ankle clonus, caused pain, as did also grasping the calf or thigh muscles. The knee-jerks were both absent, as were also ankle clonus and the Babinski response.

Expansion was good on both sides of the chest. The apex beat was not visible. The pulse was small, of poor force, and slightly accelerated. The cardiac impulse was feebly felt in the fifth interspace in the mid-clavicular line.

An area of dulness extended from the third rib to the left edge of the sternum and to one-half inch outside of the midclavicular line. The percussion note was good over both lungs and the breath sounds were clear. The liver dulness began above the fifth rib, but the organ could be felt below the costal border; the spleen could not be felt below this border.

This woman's physical and mental condition did not change much during the time she was in the hospital, except that it got worse during the last few days of her life. She continued to be helpless in her limbs and to have pain, both subjective and on handling them so as to compress the nerves and muscles.

She was put on the usual treatment for multiple neuritis, consisting of the internal use of small doses of mercury and of the salicylates, iodides, and bromides, her other treatment being symptomatic and chiefly remedies to relieve pain and produce sleep.

She died November 20, two days before her death developing great weakness, which passed into stupor.

Sections were made from the lumbar and cervical portions of the spinal cord and from the medulla oblongata and pons; also in the cerebrum from the paracentral lobules, the angular gyrus, the midprecentral, occipital, calcarine, and temporal regions. In addition, sections were made from muscle tissue and from the trigeminal, abducens, and facial nerves; also from the anterior half of the entire cerebral hemisphere. The sections from this case were stained by the Weigert hematoxylin, hemalum-acid fuchsin, Marchi, and thionin phenique methods.

The sections made from the lumbar region showed great degeneration of many of the anterior cornual cells, this being evidenced by a disappearance of dendritic processes, by peripheral displacement of the nucleus, and by central chromatolysis. The changes in the cervical cord were the same as in the lumbar region, although possibly less intense. In the gray matter of the anterior horn were seen several small hemorrhages. In this case, as in the previous one, the examination of the Weigert preparation of the anterior half of the cerebral hemisphere proved negative; the frontothalamic band and pyramidal tracts were normal.

By the Marchi method the medulla oblongata and the lumbar cord showed no typical degeneration. Sections of the medulla oblongata stained by the Nissl method showed a number of hemorrhages in the posterior part. In this locality was also found a very pronounced perivascular round-cell infiltration. There was no round-cell infiltration of the pia; it was remarkable that this perivascular round-cell infiltration within the oblongata should be so sharply confined to one or two vessels and should be so intense. At a higher level was seen a slight round-cell infiltration of the pia.

The paracentral lobules in this case seemed normal, as regards the nerve cells, but the pia was considerably thickened, and along the outer edge were found occasionally proliferation of endothelial cells forming distinct masses like those described by Spiller in a case of lead encephalopathy. The pia was in many places firmly adherent to the cortex and presented an increase in the number of nuclei. The calcarine, precentral, angular gyrus, the occipital and frontal regions presented no abnormalities.

Sections of the trigeminal nerve stained by the Marchi, the acid-fuchsin, and the Weigert methods appeared normal.

The oculomotor nerves as studied in cross-section by the hematoxylin-eosin method were normal.

Case V.—No. 266, M. T., female, white, aged thirty-two years; was admitted to the Philadelphia General Hospital (Dr. Mills' service) September 14, 1904. She denied specific disease and had had no miscarriages. She had drunk coffee in large quantities all her life, and for the past six years had been a heavy beer drinker. About six weeks before admission she gave birth to a full-term child, which died after five weeks of enteritis.

The present illness dated back to about three months previous to her admission, when she first had a numbness and tingling in her lower extremities from her knees down; shortly after this she noticed that she was gradually becoming weak and that she tired easily. After the first week of her puerperium, on attempting to leave her bed she discovered that she was unable to stand, and had pain in her lower limbs. Up to the time of her admission there had been no involvement of the upper limbs. She had been troubled with constipation for two weeks before coming to the hospital, and for the first time was catheterized on the evening of her admission. She said that her sight had been failing for six months before she was admitted.

Examination shortly after admission showed double foot-drop; both knee-jerks absent and the Babinski not obtainable on either side; an attempt to obtain ankle clonus produced acute pain; both feet were cold and the soles were somewhat moist; sensation to touch and pain was apparently normal.

Her memory for recent events was extremely poor; even though she had been in the hospital only twenty-four hours, she was unable to say how long it was since she was admitted, and she was never able to remember whether or not she had had her meals, even though she had eaten a few hours before. Two days after her admission, on being asked whether her bowels were opened that day, she replied in the negative, while her nurse had recorded two movements on her chart; again she was unable to name properly the articles of diet which she was taking, always giving the names of different foods which she had never had.

About September 27 she began to have periods of excitement, during which she would shout terrible oaths and use very vile language. On October 6 she said she had been up and around all day, and had been down town in the morning; at the same time she was scarcely able to move in the bed. She complained of much pain in her hands, but otherwise said she was feeling pretty well.

On November 7, while the interne was in the ward taking some notes, the patient said she was not able to go down town to have her pictures taken, but would go tomorrow. She talked in a mumbling or muttering manner, which could seldom be understood, and for this reason many of her delusions could not be recorded.

About September 25 she developed incontinence of urine and feces. Tenderness over the extremities had become almost unbearable. There was complete double wrist-drop, and she had become somewhat wasted both in the extremities and about the face. On November 11 the above symptoms were all present and exaggerated. On lateral excursion of the eyes nystagmus was produced.

On October 10 Dr. Shumway reported extra-ocular movements good in every direction, the pupillary reactions prompt, and that ophthalmoscopic examination showed a perfectly normal eyeground on each side.

The examination of the organs of the chest and abdomen gave no points that would be of any value in recording the nervous and mental phenomena of the case.

The muscles of the hands, especially the dorsal interossei, were slightly wasted; otherwise the upper limbs appeared well developed. Motion was present in all directions; resistance to passive movements was much decreased in both upper extremities; wrist-drop was present on the left side, not on the right. The grasp of both hands was diminished, slightly more so on the left side. The biceps, triceps, and wrist reflexes were absent on each side. Pressure over the course of the nerve trunks of both upper limbs caused pain, and sensation to touch and pain appeared to be about normal in both.

Neither lower extremity showed any atrophy, and motion was greatly lessened in both, the patient being able only to bend the knee to a small angle, and unable to raise the feet from the bed.

The patient became weaker, both physically and mentally, with, however, occasional short periods of slight improvement. She developed a bed sore on the left hip, the incontinence of urine and feces became more marked, the pain on pressure and manipulation of nerves and muscles persisted, and even when she became apathetic to the point of stupor, was sufficient to arouse her with evidences of distress.

A tendency to excitement was noted on one or two occasions. Several records were made of extreme tenderness of the muscles and also of tenderness and pain in the limbs, Small swellings appeared on the arms, the forearms, feet, and other places, these apparently being connected in some way with the inflammation of the nerves. Her temperature rose and fell several times. The day before her death the temperature, pulse, and respirations were all increased. The lungs were full of coarse râles. She died December 31.

Sections were made from the lumbar and cervical portions of the spinal cord, and from the medulla oblongata and the pons. In the cerebrum sections were made from the paracentral lobules, the angular gyrus, and the midprecentral, occipital, calcarine, and temporal regions. Sections were also made from pieces of muscle, and from the external plantar, the sciatic, the internal plantar to the great toe and the pneumogastric nerve. In addition, sections were made of the anterior half of the entire cerebral hemisphere.

These various sections were stained by the thionin phenique, Weigert hematoxylin, hemalum-acid fuchsin, and in some cases the Marchi methods.

Sections from the lumbar region showed considerable chromatolysis of the nerve cells of the anterior horns, with peripheral displacement of the nuclei. This chromatolysis seemed to invade the whole of the cell body. Some of the cells were greatly vacuolated. In three serial sections from the midlumbar region, an accumulation of round cells was seen lying immediately in front of and mesial to one of the anterior horns. These cells were both polymorphonuclear and mononuclear in type. In the midst of this accumulation was seen a dense body, staining with thionin dark blue-black. Under the oil immersion objective, this was found to consist of an accumulation of cocci. Near the periphery of this dense mass was seen what suggested diplococci. A blood vessel could be seen running from the periphery of the cord toward this cellular infiltration.

The cervical region showed about the same alterations as regards the anterior cornual cells, with the exception that the change was not so pronounced. The white matter of the lumbar and cervical regions showed no degenerations by the alum-hematoxylin and acid-fuchsin methods. Neither was there any system degeneration as shown by Weigert's method, the columns of Goll, Gowers' columns, the direct cerebellar tracts, the crossed and direct pyramidal tracts, etc., being particularly examined.

In the paracentral lobules were small accumulations of cells, both polymorphonuclear and mononuclear in type, which were apparently not in relation with bloodvessels, although it is possible that bloodvessels might be found leading to this accumulation in sections either immediately above or immediately below the area examined. Some very small, deeply staining cocci, like those noted in the lumbar cord, were also found in these infiltrations. Some of the cells of Betz were much degenerated and showed central chromatolysis and peripheral displacement of the nucleus.

In the right temporal region there were also found collections of polymorphonuclear and mononuclear cells, as noted above. In this region there were associated close aggregations of red blood corpuscles, together with the same cocci-like bodies.

In the right calcarine region, some of the bloodvessels of the pia, cut transversely, showed in addition to the thickly packed erythrocytes, a goodly number of polymorphonuclear cells and cocci-like bodies within the lumen.

The right angular gyrus, the right mid-precentral, and the right occipital region appeared normal.

In the muscle the connective tissue was greatly increased, both between the muscle bundles and between the individual muscle fibers. The muscle fibers were much atrophied. The small nerve fibers within the muscle were much degenerated. The external plantar nerve was greatly degenerated, and the connective tissue about it was thickened. The sciatic nerve was intensely degenerated, as were also the pneumogastric and the internal plantar to the great toe.

The Weigert hematoxylin preparation of the anterior half of the cerebral hemisphere showed no degeneration of the frontothalamic band in the anterior limb of the internal capsule, or of the pyramidal tracts in the posterior limb.

Case VI.—No. 431, W. B., male, white, aged seventy years; was admitted to the Philadelphia General Hospital (Dr. Spiller's service) January 27, 1908.

On account of his mental condition it was impossible to obtain any satisfactory statement of past illnesses, but it is probable that his condition on admission dated back about a year. He acknowledged great alcoholic excess, and gave his name and age readily, but when questioned as to his whereabouts, he said he was in the penitentiary, that he had been placed there by the port warden for "trifling with liquor," that he had known the examining physician for twenty years, and that he had been in that particular ward one month (this, six hours after admission).

At times his replies had no relation to the question asked. There was no defect in speech.

The pupils were contracted, the left much more so than the right, the right being irregularly oval in outline. The irides reacted promptly but slightly to light, but owing to the myotic pupils the amplitude of contraction was not great. They also reacted fairly to accommodation and convergence.

There was no ptosis nor nystagmus, nor was there any extra-ocular paresis. The other cranial nerves were normal.

The upper limbs showed a diminution in the strength of his grip, especially in the right hand. He moved them in all directions freely and presented no ataxia with eyes open or closed. Resistance to passive motion was fair. There was no muscular atrophy, wrist-drop, or contractures. The biceps tendon reflex was prompt on the left but diminished on the right. The triceps tendon reflex was diminished on both sides.

Examination of the lower extremities showed normal range of motion, flabby musculature, tenderness of muscles and over nerve trunks on pressure. No ataxia on heel to knee test, absence of patellar tendon and Achilles tendon reflexes in each limb. There was no Babinski reflex.

He could not walk without looking at the ground, and when he attempted to do so would stagger.

The pain in his lower limbs is worse at night. He complained that his eyesight was failing. He had incontinence of urine. Pressure in lumbar region and along the spinal column elicits tenderness.

The patient's weakness increased, his mentality became more clouded, and he died February 20, 1908.

Sections of the spinal cord in the lumbar and cervical enlargements show no abnormality of the white matter, but there is a pronounced axonal chromatolysis in the anterior cornual cells, more severe on the left side. Pieces of nerve and muscle, unfortunately not labelled as to name at the autopsy, show pronounced degenerative change.

Case VII.—P. F. H., male, white, aged forty-six years; was admitted to Dr. Spiller's service at the Hospital of the University of Pennsylvania, September 27, 1908.

He is a train despatcher by occupation, and has not been exposed to cold or wet weather.

He denied syphilis and has used alcohol in moderation, taking on an average of one drink of whiskey and one bottle of beer per week.

His trouble began thirteen days before his admission, at which time he complained of weakness in his left foot. The next day, on awakening, his left lower limb felt numb below the knee. In twenty-four hours this numbness involved the whole left lower limb. In two or three days the numbness had invaded the right lower limb about the knee. Two days after this the right upper extremity became weak, and twenty-four hours afterward the right lower limb, previously numb, became weak. A few days after some weakness developed in the left upper limb.

On examination, considerable power was evidenced in flexion at the right ankle, also in flexion and extension of the right big toe. The other toes of the right foot showed but little motion. He could draw up left lower limb slightly at hip, otherwise all motion in this extremity was abolished. The patellar tendon and Achilles tendon reflexes were absent on each side. There was no movement on plantar stimulation. Sensation for touch, pain and temperature was perfectly preserved in the lower limbs. The cremaster reflex was absent on the right but present on the left. The abdominal reflexes were not distinct. The muscles of the lower limbs were tender to pressure.

On September 28 he developed a dull aching pain in his lower limbs.

On September 30 it is recorded that the grasp of each hand and resistance to passive motion in upper limbs are only slightly diminished. He was unable to elevate the right upper limb at the shoulder.

Some slight tenderness on pressure was present in muscles of the upper limbs. The triceps and biceps tendon reflexes were absent on both sides.

On October 13 it is recorded that the patient had a severe chill "from head to foot," in which he said he did not feel cold and during which he was in a "profuse warm sweat." During the three days following he had a number of these "chills," and on October 16 Dr. Spiller recorded: "The upper limbs are distinctly weaker in all parts than they have been. He cannot elevate either very well at the shoulder, nor can he readily flex either limb at the elbow. This increased weakness of the upper limbs has developed since the chills began three days ago."

On October 14 twitching of the muscles of the right lower limb is recorded. The muscular twitching gradually increased in extent and severity. On October 20 his "mind began to wander." From this date until November 30 he was frequently irrational, at times having falsification of memory, at times his orientation being markedly defective. There is no record of any psychic symptom after November 30.

On October 26 Dr. Spiller made the following note: "The upper limbs have been paralyzed for some time except in the hands. The muscles of all the limbs are still very painful to touch. He has had jerkings of all the limbs for some time, especially in the upper limbs. He is somewhat confused, and said this morning, 'Tell those men to go up the road.' He has had frequent chills involving all parts of the body."

From October 26 a gradual but at times, interrupted improvement took place and on February 14, 1909, it is recorded that he was sitting up on a chair about two hours a day and was steadily gaining in strength.

Case VIII.—No. 274, L. McC., male, white, aged forty-nine years; was admitted to the Philadelphia General Hospital (Dr. Spiller's service) October 17, 1904.

The family history was negative. He has had diseases common to childhood but there is no mention of venereal disease in his record. He used alcohol in excess from twenty-one years of age, and was frequently "dead drunk."

He gave a history of two attacks of delirium tremens.

For six years prior to admission he drank heavily. The last two weeks of this period he had marked tremor and during the last week anorexia and insomnia.

On admission he complained of pain in his epigastrium and left chest, and had hallucinations of the sense of touch. He was running a temperature not unlike malaria, and there was great tenderness to pressure over nerve trunks.

Notes made on December 28 record that the motor cranial nerves were normal, but that there was very distinct tenderness on pressure over the exits of the infra-orbital and supra-orbital nerves of both sides. The irides reacted sluggishly to light, accommodation, and convergence. There was no loss of tactile or pain sense in the face. There was present a fine tremor of both hands—involving the hand as a whole. The power of extension of the carpus was very weak on each side, but there was no distinct wrist-drop. There was great wasting of the interosseous spaces of both hands. No loss of the tactile or pain sense in either upper limb, and the extensor surface of each forearm was hyperesthetic. The biceps tendon reflexes were greatly decreased. There was pain on pressure over the large nerve trunks in the upper limbs. The lower extremities were greatly wasted, particularly the inner surfaces of the thighs and the dorsum of both feet. The patellar tendon reflex was greatly decreased. There was no loss of sensation in the lower limbs but there was much tenderness to pressure over the nerve trunks.

The sphincters of the bladder and rectum were well controlled.

On January 10, 1905, it is noted "patient still complains of pains in the arms and legs which are beginning to show signs of contractures." The extremities were splinted to counteract their contractures, and on January 15 it is recorded: "The extremities have been splinted daily and are now straighter than formerly, and he seems to have less pain and more use of them."

The patient died January 22, 1905, the immediate cause of death being a laryngeal edema.

Examination of sections from the paracentral lobule stained by the Weigert hematoxylin, thionin phenique, and hemalum-acid fuchsin methods show no abnormality with the possible exception of a hyperpigmentation of the cells of the Betz. Sections from the lumbar and cervical enlargement were stained by the Weigert hematoxylin, hemalum-acid fuchsin, and thionin phenique methods. There was much pigmentation of the anterior cornual cells. Occasionally there could be found a cell with eccentric nucleus and central chromatolysis, but in the large majority of cells the tigroid substance stained well. With the exception of the fact that there seemed to be many spaces from which axis cylinders and sheath had disappeared, the white matter was normal. Sections of the medulla oblongata stained as above, proved normal.

Nerves from the feet were stained by the Weigert hematoxylin and hemalum-acid fuchsin methods, and teased Marchi preparations were likewise made; by all these methods intense degeneration was noted.

Sections of muscle stained with osmic acid did not show any of the typical osmic acid reactions seen in cases where degeneration is moderately advanced. But the hemalum-acid fuchsin preparations of muscle showed a beginning Zenker's hyaline degeneration often noted in severe intoxications, such as typhoid fever, high grade anemias, etc.

Case IX.—No. 282, K. H., female, white, aged forty-two years; was admitted to the Philadelphia General Hospital (Dr. Spiller's service) February 21, 1905, when the following history was elicited:

Her father died suddenly at forty-five years, cause unknown. Her mother is alive and well at fifty-four years. Five sisters are living and well. One sister died in confinement. The patient is sure that her mother had had no miscarriages or still-births.

The "milder" diseases of childhood and typhoid fever at fifteen years are recorded. She also gave a history of acute articular rheumatism in 1900

She was married at nineteen years of age, but lost her husband through an accident, having had no issue by him. She married again at thirty-five years of age, and had one healthy child, which died at one year of membranous croup, and one still-born child. She never had any miscarriages. Since her early childhood she has been accustomed to a glass of beer every morning and at night upon going to bed. At times her morning potation would be varied by whiskey in her tea.

She spent the night of January 1 in drinking, although she insists she was not drunk. Next morning she had a great deal of nausea, with pain and weakness in her legs from her knees down. This pain and weakness increased, and for a number of days prior to her admission she was incapacitated, spending her days on a sofa. All this time she was taking her full quota of alcohol.

On her admission it is noted that she had no idea where she was, did not remember how long she had been sick, and had no idea of time.

She heard voices talking to her, and mistook the strangers about her for friends. Her pupils were larger than normal, the right being slightly larger than the left. The reaction to light was not noted, and as she could not obey commands, it was impossible to tell whether her irides reacted to accommodation and convergence.

Her fifth and seventh nerves were uninvolved and her tongue protruded in the median line.

The examination of her upper limbs showed that her grip was weak, especially in the left hand. The development was poor with a suggestion of atrophy in the forearm and hands, particularly on the palmar surface. Motion was free in all directions. There was no tenderness on pressure, and sensation for touch and pain was normal. No fibrillary tremors were present. There seemed to be some ataxia, possibly more in the right arm. The reflexes of the arms could not be elicited, because she would not relax, but there was no spasticity.

Her lower limbs were very paretic, the paralysis of the peroneal group being greater on the left than on the right. Dorsal flexion of the right foot was possible, but not of the left. There were areas of discoloration on each leg below the knee, having the appearances of old hemorrhages beneath the skin. The patellar and Achilles tendon reflexes were lost on each side. The sensation for touch and pain was distinctly impaired in both limbs below the knee. The muscles of the calf were tender to pressure in both limbs. Irritation of the soles of the feet gave no motion of the toes.

Her general condition gradually became worse. She was irrational, at times noisy, at times quiet. She had incontinence of bladder and rectum.

She died March 12, 1905.

Sections from the paracentral lobules were stained by the Weigart hematoxylin and thionin phenique methods, and showed thereby no abnormality except a slight small, round-cell infiltration in the pia.

The eighth, ninth, tenth, and eleventh cranial nerves were studied, and presented no abnormality with the exception of a slight small, round-cell infiltration in the sheath of the eighth nerve.

Sections of the spinal cord were made in the cervical and lumbar enlargement, and also in the mid-thoracic region. These were stained by the Weigert hematoxylin, hemalum-acid fuchsin, Marchi, and thionin phenique methods, and the following conditions noted. There was present a degeneration of the posterior columns, which in the lumbar enlargement completely fills the space between the two posterior cornua and is of pronounced intensity. At the mid-thoracic level it is seen that the degenerated and the normal nerve fibers of the posterior columns are about equal in number, with a tendency for the degenerated fibers to pass toward the median septum. In the cervical enlargement the appearance is fairly diagrammatic, in that the fibers immediately next to the median septum are preserved, those midway between the median septum and posterior horn on each side are degenerated, and those next to the posterior horn are normal.

The white matter of the root of the spinal cord is normal.

The anterior cornual nerve cells in the cervical enlargement are in normal condition. Those in the lumbar enlargement are very greatly degenerated—the degeneration being axonal in type with here and there vacuolation.

There is an intense, small, round-cell infiltration of the pia of the cord most pronounced in the lumbar and cervical regions.

Teased Marchi preparations were made of the pneumogastric nerve, as well as a nerve from the sole of the foot, and in both was an intense degeneration shown.

The pneumogastric nerve was also stained by the Weigert hematoxylin and hemalum-acid fuchsin methods, and not only was the degeneration most marked, but also the great distention of the bloodvessels throughout the substance of the nerve.

Muscle from the foot showed intense degeneration by the Marchi method, the muscle fibers appearing as a mass of the finest black dots.

Case X.—No. 84, P. S., male, white, aged forty-eight years; was admitted to the Philadelphia General Hospital (Dr. Spiller's service) January 16, 1902, presenting a history from which I note a few relevant observations as follows: He was born in Ireland. His father and mother, six brothers, and three sisters are living. One brother died of typhoid pneumonia. There is no note as to venereal disease. He has had at different times fractures of his right arm, fingers of the left hand, three ribs on the right side, the right leg, and three toes of the left foot. No two fractures occurred at the same time. There is a history of "chills and fever" sixteen years ago. For the past twenty years he has worked in lead.

Four weeks prior to admission he began having steadily increasing pain in his back and abdomen, the latter being decidedly sensitive on palpation.

On admission he was unable to flex either of the upper extremities at the elbow joint, but there was some power of pronation and supination. Double wrist-drop as well as wasting of the muscles of the hands and forearms are noted. Sensation in the upper extremity was normal and the reflexes were absent.

Since his admission he has suffered a total loss of power in his lower extremities, with abolition of reflexes with the exception of the plantar reflex, which is normal.

Sensation in the lower extremities was normal.

Delirium, incoherence, mental dulness, and "uremic symptoms" are all noted.

The examination of his eyes showed a catarrhal condition of conjunctivæ with a mucopurulent discharge. The pupils were 3 mm., and the irides reacted very sluggishly to light, particularly the right iris. A high grade neuroretinitis without hemorrhage was noted in each eye but more pronounced on the right.

From February 10 to the time of his death on March 19, 1902, incontinence of bladder and rectum is recorded.

Sections from various regions of the central cortex were studied by the hemalum-acid fuchsin and Weigert hematoxylin methods and showed nothing abnormal. The cerebellum also proved normal.

A section from the medulla oblongata in the mid-olivary region showed a number of swollen axis cylinders in the motor tracts of each side by the hemalum-acid fuchsin method.

Sections of the spinal cord at the eighth cervical segment and in the middle of the lumbar enlargement were stained by the Marchi, hemalum-acid fuchsin, and thionin phenique methods. The following changes were noted: Swollen axis cylinders in the cross pyramidal tracts, osmic acid degenerative reaction in the posterior nerve roots immediately within the cord, as well as in the strands of fibers, of the anterior nerve roots passing from the periphery of the cord into the anterior cornua, intense osmic acid degenerative reaction of the anterior cornual cells, axonal degeneration of the anterior cornual cells of very intense degree, the only group spared being that most anterior and medial.

Marchi preparations of the posterior roots and root ganglia show intense degeneration of both nerve fibers and ganglion cells.

The anterior and posterior nerve roots of the sacral, lumbar and cervical regions show great degenerative change by the Marchi and the hemalum-acid fuchsin methods.

The right and left sciatic and right and left median nerves were stained by the Marchi, Weigert's hematoxylin, and hemalum-acid fuchsin methods, and pronounced degenerative changes found in all.

A piece of the left pronator radii teres and of a muscle in the anterior aspect of the right forearm showed pronounced degeneration by the Marchi and hemalum-acid fuchsin methods.

Sections of the left optic and left oculomotor nerve proved normal.

Case XI.—No. 156, D. F., male, white, aged forty-two years; was admitted to the Philadelphia General Hospital May 11, 1900.

Although he was in the hospital for over five months yet the notes are meagre in the extreme and many interesting data are wanting.

The family history was negative. As to the patient's past history, it is recorded that he had "fever and ague" fifteen years prior to admission, also that he had been a heavy drinker for fifteen years, but had never had delirium tremens. He acknowledges gonorrheal infection.

The account he gave of his condition immediately prior to admission is that he had been drinking heavily for six weeks, at the beginning of which time he began to suffer sharp pains in both thighs.

In a week his thighs were tender on pressure and his lower limbs began to get "weak and stiff." This condition of affairs has been steadily increasing.

On admission there was noted copper-colored scars on his legs; also white scars of a punched-out appearance. He could move his lower limbs, but he complained of pain in the calves and thighs. The patellar tendon reflex was absent. There was no anesthesia for touch demonstrable. All that is noted concerning the upper limbs at the time of admission is that there was no pain over the median nerve on pressure and that there was no extensor paralysis of either hand. It is probable that the upper limbs at this time were uninvolved.

On October 1 it is recorded: "The patient is absolutely confined to bed and practically unable to move, especially his lower limbs. He complains of great pain in legs." This rather implies that the paralysis had by this time invaded the upper limbs.

On October 10 there is noted: "Has had incontience of urine and feces."

He died October 16, 1900.

Sections were made from the mid-olivary region and the motor decussation of the medulla oblongata, from the lumbar enlargement of the spinal cord, transversely through the lumbosacral plexus, median, musculospiral, and sciatic nerves and also of muscle.

The medulla oblongata proved normal.

The white matter of the spinal cord showed no swollen axis cylinders, but there were many spaces in both the lateral and posterior columns from which axis cylinders and their medullary sheaths had disappeared.

There was a moderate number of cells in each anterior horn showing axonal degeneration.

All the nerves examined showed degeneration, the lumbar much more so than the brachial. The muscle showed a very moderate amount of osmic acid degenerative reaction.

Case XII.—No. 106, J. G., male, white, aged fifty-two years; was admitted to the Philadelphia General Hospital (Dr. Mills' service) December 26, 1901.

He had had syphilis twenty years prior to this date. He was well up to December 24, two days before admission. During the day and night of December 23, he drank heavily sitting up all night long in a chair. The next morning he got up from the chair, and in attempting to walk to his bed fell to the floor, where he remained all day, suffering severe pain as well as motor paralysis in each lower extremity.

On admission it was found that the pain in the right lower extremity was chiefly sciatic in distribution and in the left was confined to the foot. The left lower extremity also showed slight swelling and cyanosis, and its temperature was distinctly warmer than on the right. Both lower limbs could be moved, but the loss of power was greater in the left, which could not be raised from the bed. The tendon reflexes of the lower limbs were increased on the right and absent on the left, with the exception of the plantar reflex, which was normal on each side. There was anesthesia, thermo-anesthesia, and analgesia on the anterior aspects of the left lower limb from the knee to the upper third of the thigh, as well as partial analgesia below the knee. There were no sensory changes in the right lower extremity except hyperesthesia and pain on pressure along the course of the sciatic nerve. Pain on pressure in abdomen and thorax.

There was no involvement of upper extremities, face, or eyes. He retained control of his bladder and rectum until the day of his death, December 30, four days after his admission. On this day his mental faculties were clouded; he did not move his upper limbs on command—whether because he did not comprehend or because of motor paralysis could not be determined. On this date it is recorded that both biceps-jerks and knee-jerks were absent. The death was due "apparently to respiratory paralysis."

Sections were prepared from the cervical and lumbar enlargements, and were stained by the Weigert hematoxylin, hemalum-acid fuchsin, and thionin phenique methods. There were a few unmistakably swollen axis cylinders in the crossed pyramidal tract of each side in the cervical region, but none were found in the lumbar region. The anterior cornual cells of the cervical enlargement were in a fair state of preservation. In the lumbar enlargement there was extensive axonal reaction of not very pronounced type.

The vagus, left popliteal, and right and left plantar nerves were teased and stained by the Marchi method. The left plantar and the vagus showed degenerative changes. The left popliteal and the right plantar were negative.

Case XIII.—No. 20, R. D., male, white, aged twenty-nine years; was admitted to the Philadelphia General Hospital (Dr. Potts' service) June 8, 1902.

His history records him an Italian laborer, and states that on June 2, six days before his admission, he was in perfect health. On awakening in the morning of June 3 he had pain in his right upper limb, followed in a short time by paralysis of the same. Within forty-eight hours he had become completely quadriplegic, the order of paralysis being right upper limb, left upper limb, right lower limb, left lower limb. The limbs were tender to pressure, especially over nerve trunks.

The pupils were semidilated, the left being larger than the right. Both irides reacted to light, accommodation, and convergence.

The tongue was protruded in the median line.

All tendon and muscle reflexes were uniformly absent. He had control of his sphincters.

There was no loss of tactile sensation. He could slightly move his right thumb, and rotation of his thighs and legs was possible to the extent of about a quarter of an inch.

There was no history of alcohol, venereal disease, nor was there any history of acute or infectious disease.

He died June 10, two days after his admission.

Sections were made from the cervical and lumbar enlargements and stained by the Weigert hematoxylin, hemalum-acid fuchsin, and thionin methods.

Swollen axis cylinders were found in the crossed pyramid tracts, but not in the posterior columns.

The anterior cornual cells present a diffuse and intense chromatolysis in both lumbar and cervical regions. In only a few cells could it be said that the chromatolysis was axonal in character. In one of the Weigert preparations examined from the lumbar enlargement a right posterior root happened to be cut longitudinally, entering the cord. Its myelin sheaths stained well. Marchi and Weigert preparations of the sciatic nerve demonstrate a moderate degenerative change.

Case XIV.—No. 237, J. A., male, white, aged sixty-seven years; was admitted to the Philadelphia General Hospital (Dr. Spiller's service) July 16, 1904, at which time the following history was noted:

He denied venereal disease, but had had typhoid fever, as well as "ordinary diseases of childhood."

Since fourteen years of age he has been a painter. In 1876, when about thirty-nine years of age, he began having attacks of weakness in his upper extremities, having in all six or seven up to the present time.

His present trouble began about two weeks before his admission, and was characterized at first by diarrhea and loss of power in his upper limbs. For the week prior to admission his stools were bloody. He had no pain anywhere.

His ward notes record him as being cachectic in appearance and somewhat edematous in the face. His pupils were equal and the irides reacted sluggishly. There was no blue line on the gums. The bloodvessels did not show much sclerosis. A bilateral wrist drop with decided atrophy of the thenar and hypothenar eminences was present, and a marked intention tremor in both hands was demonstrated. The shoulder muscles of both extremities were uninvolved, and he could elevate voluntarily his arms above his head.

The biceps tendon reflex was weak on the left side, and uncertain on the right. The triceps tendon jerk could not be obtained on either side. Although there was atrophy of the muscles of the arms, yet the wasting was not so great as in the forearms. The extensors on the back of the forearms seemed to be more involved in the atrophy than the flexors.

In the lower extremities there was paralysis with atrophy in the peroneal group on each side. The lower limbs were flaccid, except at the ankles, and without movement, although he seemed to have retained some power in each thigh. The feet were in the position of talipes equinovarus, and could not be straightened. The patellar tendon and Achilles tendon reflexes were absent on each side. The Babinski reflex was uncertain on right, and could not be tested for on the left, owing to an amputation at the metatarso-phalangeal articulation.

Some days before his death he developed a dysentery from the exhausting influence of which he rapidly sank.

Notes made on the day of his death record incontinence of bladder and rectum; this was in all probability due to exhaustion and the fluid condition of his feces rather than to any disturbance of the sphincter nervous mechanism.

Sections of the right and left paracentral lobules stained by the thionin phenique method showed a very distinct chromatolysis of the large Betz cells. This degeneration was axonal in character and occasionally very severe, the nucleus being displaced to the periphery of the cell and only a small rim of tigroid substance being preserved.

Sections of the spinal cord from the cervical and lumbar enlargement as well as the midthoracic region were stained by the Weigert hematoxylin, hemalum-acid fuchsin, thionin phenique, and Marchi methods.

Swollen axis cylinders were found in the lateral and posterior columns, and also in the entering posterior nerve roots. The anterior cornual cells were moderately degenerated, many being in a normal state. The degeneration was of the axonal type, and greater in the lumbar region than in the cervical, and more pronounced on the left side of the cord.

Teased preparations from the left brachial plexus and from the right ulnar nerve were stained by the Marchi method, and showed intense osmic acid degenerative reaction, which finding was also borne out in the Weigert hematoxylin sections.

Sections of voluntary muscle stained by the hemalum-acid fuchsin method show only a moderate amount of degenerative change, many fields appearing perfectly normal.

The optic, oculomotor, fourth and fifth nerves of both sides proved normal, as did the Gasserian ganglia.

Case XV.—No. 441, J. R., male, white, aged sixty-four years; was admitted to the Philadelphia General Hospital (Dr. Spiller's service) January 10, 1908.

His family history was negative. He had always used great quantities of tobacco. He had been using alcohol for the past forty years, averaging about two glasses of whiskey and a pint of beer daily. He denied specific infection.

During the early part of November, 1907, he began to be troubled with weakness in his lower limbs. By Christmas he developed sharp pains in his calf muscles, which grew steadily worse. His feet were swollen. For two weeks prior to his admission his lower limbs had become numb, and he was unable to walk unassisted. Also at this time he first noticed that his left hand was becoming weak. This was followed shortly by weakness of the right hand. Both hands were numb during the week preceding admission.

Examination of his cranial nerves discovered no abnormality. Examination of the upper limbs showed that the muscles of both shoulder girdles and both forearms were distinctly wasted. There was great weakness of the extensor group of each forearm, on the left amounting to a wrist drop.

The grip in either hand was very weak, and the resistance to passive motion was very greatly diminished. The biceps tendon and triceps tendon reflexes on the right were absent, on the left were just barely perceptible.

The lower extremities showed bilateral foot-drop, flabby musculature, feeble resistance to passive motion, and marked tenderness along all nerve trunks. No ataxia was demonstrable. The patellar tendon reflex was absent on the left and barely perceptible on the right. Both Achilles tendon reflexes were absent.

There was no sensory disturbance found, and stereognostic perception was preserved. The bladder and rectum were normally controlled.

On February 24 he had a number of convulsions with unconsciousness.

By February 29 a large bed sore had formed on his back.

There was no abnormality noted in the ophthalmoscopic examination of his eyegrounds.

He died March 3, 1908.

Sections from the right and left paracentral lobules were stained by the Bielschowski, thionin phenique, hemalum-acid fuchsin, and hematoxylin-eosin methods, and nothing abnormal was found.

Sections from the eighth cervical, sixth thoracic, and third lumbar segments were also stained as above with the addition of the Weigert hematoxylin method. They showed pronounced degeneration in the anterior cornual cells axonal in type. No swollen axis cylinders were found in the white matter of the cord.

A piece of muscle with nerve attached was taken from the arm at necropsy and both nerve and muscle showed pronounced and well-advanced degeneration by the Marchi and hematoxylin-eosin methods.

Case XVI.—No. 354, N. F., female, white, aged twenty-three years; was admitted to the Hospital of the University of Pennsylvania (Dr. Mills' service) October 18, 1906. Her past history is not relevant.

She had been married for five years and had had two children and no miscarriages.

Her trouble on admission dated back about ten weeks, and began with nausea and an inability to retain food. At this time, about the second week in August, she was between six and seven months pregnant.

To the nausea was added fever and a gradual loss of power in her lower limbs. She also had what she called "rheumatic pains" in her abdomen.

On account of the severity of the symptoms labor was induced on October 1, at the eighth month of pregnancy, and she was delivered of her child by forceps. For two or three days following she improved, but in a short time began to get worse again, having fever, chills, and delirium. An exacerbation of her symptoms the day before caused her admission as noted above. The excretion of urine had gradually diminished, and on admission she had not voided any since the evening of the day before.

On admission she was delirious, with a temperature of 103° F., a pulse of 120, and respirations 40.

In her initial examination her irides reacted to light, accommodation, and convergence. There were no ocular palsies, but some lateral nystagmus.

The upper extremities showed marked ataxia with athetoid movements of the fingers when the attention was attracted. There was flexion of the carpus with overextension of the phalanges.

There was much atrophy of the upper limbs, thenar and hypothenar eminences, associated with loss of power. There was also pain on pressure over the nerves.

The lower limbs were very much atrophied. There was absence of patellar tendon reflex on either side, no ankle clonus or Babinski reflex. There were "rheumatic" pains in the lower limbs and abdomen, with great tenderness on pressure.

The rectal and vesical sphincters were controlled. She gradually lost ground, and died October 22, four days after admission.

It is worthy of note that on the two prior pregnancies she had much the same symptom complex as above stated, but the difficulty cleared up after delivery.

Sections of the spinal cord at the mid-thoracic level and of the second lumbar segment were stained by the hemalum-acid fuchsin method, and upon examination showed swollen axis cylinders in the cross pyramidal tracts of each side. The Marchi method failed to show any degeneration, the change in the myelin not having progressed far enough to cause the osmic acid reaction. Sections from the lumbar enlargement stained by the thionin phenique method showed unmistakable, though not very advanced, changes in the anterior cornual nerve cells. Where the chromatolysis is at all pronounced it is axonal in character.

Teased preparations of the vagus, plantar, and posterior tibial nerves were stained by the Marchi method and showed intense degeneration. The same nerves stained according to Weigert's hematoxylin and the hemalum-acid fuchsin methods likewise showed great change.

Unfortunately the cervical enlargement was not removed at the necropsy.

I shall now detail the history and pathological examination of a case which might easily have been mistaken for one of brachial plexus neuritis.

Case XVII.—J. B., male, white, aged sixty-six years; was admitted to the Philadelphia General Hospital on January 15, 1902.

The family history was negative. He was a salesman, and was born in England. A short time before the present trouble began he had been engaged in addressing envelopes.

So far as his past medical history is concerned, he had used alcohol freely for years. He denied syphilis.

Three years prior to his date of admission he had an attack in which he was aphasic for two weeks. When it is taken into consideration that the loss of speech was only temporary, and that no trace of a brain lesion was found after death to account for it, also when his advanced age be noted, it is not at all improbable that the aphasia was uremic in character. Two months ago some weakness of the right hand occurred, which progressed to a paralysis which in time slowly ameliorated.

About the first of January, 1902, he fell on the ice, striking his right shoulder. There was some ecchymotic discoloration but no loss of function at the time. Eleven days elapsed before the right upper limb showed any change, at which time it appears, from the very incomplete history, that a weakness suddenly developed. The examination on admission—three days after the development of his monoplegia—showed both knee-jerks slightly exaggerated, but no ankle clonus.

The movement at the right shoulder-joint was very limited, but was fairly good at the right elbow-joint. He was able to move his fingers fairly well and the position of his arm at rest was natural, with slight flexion of the forearm.

His death occurred on July 11, 1902. Unfortunately there is no record of the immediate antemortem condition.

Sections were made from the upper cervical region, eighth cervical segment, and mid-thoracic region, and were stained by the Weigert hematoxylin, hemalum-acid fuchsin, and Marchi methods.

In the eighth cervical and first thoracic segments there were seen in the right lateral column, adjoining the anterior horn, sclerotic vessels extending from the periphery of the cord. There was also proliferation of neuroglia tissue surrounding the bloodvessels near the right anterior horn, and the walls of the bloodvessels showed great thickening by the hemalum-acid fuchsin stain. The anteroposterior diameter of the right anterior horn was much less than the left, and the lateral and median portions showed a great reduction in the number of the medullated nerve fibers, the lateral portion containing hardly any. In the lateral region of the anterior horn the nerve cells had entirely disappeared.

Sections from the upper cervical and midthoracic regions appeared normal.

The Marchi method showed no degeneration.

I have omitted quoting the general findings at the autopsies as being mostly irrelevant to the subject under discussion, but the following points seem to be of interest as possibly indicating toxic conditions, existing some time prior to death:

Tuberculous process was found in Cases V, VIII, and XI.

Chronic interstitial nephritis was found in Cases I, II, V, VI, X, XI, XII, XIV, and XV.

In Case XIV there was "dysentery" for some time prior to death, and in Case VI there was "carcinoma of the stomach."

Alcohol was a factor in Cases III, IV, V, VI, VIII, IX, XI, XII, and XV.

Lead was a factor in Cases II, X, and XIV.

Pregnancy and the puerperium were factors in Cases IV, V, and XVI.

Déjerine, in 1881, described peripheral neurotabes (tabes ou nervo-tabes périphérique). One of the main diagnostic points which he considered as being against this diagnosis was the presence of the Argyll-Robertson pupil.

In speaking of tabes he says:[2] "Westphal, Pierret et moi-même nous avons insisté sur la fréquence des altérations des nerfs cutanés chez les tabétiques et j'ai cherché à montrer la part que jouent ces névrites périphériques dans la production des troubles de la sensibilité cutanée, si fréquents chez les tabétiques, ainsi que leur rôle probable dans la physiologie pathologique de l'incoordination."

Eichhorst[3] not only speaks of the degenerative changes in the cutaneous nerves in cases of tabes, but also says that "die Degeneration setzt sich auch auf die grösseren Nervenstämme fort."

Now, Case I presented in life the appearance of tabes dorsalis. She had a perfectly developed Argyll-Robertson sign in both eyes. Examination of the spinal cord discovered no abnormality, but in the nerves and muscles the diagnosis of multiple neuritis was positive.

This case in my opinion makes the absence of the Argyll-Robertson symptom as a *sine qua non* in peripheral pseudotabes not tenable.

Oppenheim, cited by Remak,[4] found in a case of alcoholic multiple neuritis "Pupillenstarre (links nur reflectorisch, rechts auch bei convergenz)."

In the consideration of the Argyll-Robertson pupil I do not purpose to discuss the different theories as to the conduction pathway from the retina to the anterior colliculli and out again via the oculomotor nerve. I shall accept as probable that the stimulation arising in certain cells of the retina passes backward by way of the optic nerve, that a partial decussation takes place at the chiasm and that the first link in the chain is completed in the lateral region of the anterior colliculli. From here a new system of neurones conducts the impulse to the oculomotor muscles of the same and the opposite side, and that another relay of neurones forms an efferent light reflex path via the oculomotor nerve, which terminates in the ganglion cilliare as its motor root.

According to the conception of a reflex, should this arc be broken in any part of its extent the reflex would cease and determine.

Now, the Argyll-Robertson pupil in tabes Marina considers to be due to degeneration of cells in the ciliary ganglion.

Ludwig Bach[5] takes issue with his hypothesis on the ground that the cells of the ciliary ganglion are not only normally variable in appearance, but that decided post-mortem changes (schwere Veränderungen) take place soon after death.

It has been, of course, impossible for me to follow in my microscopic study the entire reflex arc of the three cases having Argyll-Robertson pupils, but it can be definitely said that the optic nerves presented no abnormality, which makes it presumptive that the first link in the chain—*i. e.*, from the retina to the end station in the anterior colileulli—played no part in the breaking of the reflex arc.

The ciliary ganglia were not accessible to me for study, and the axonal type of degeneration found in some of the cells in or near the third nucleus cannot be weighed as evidence on account of the impossibility of saying definitely that they belonged to the oculomotor system. My finding, therefore, of many swollen axis cylinders in the oculomotor nerves of the cases with Argyll-Robertson pupil is in no way decisive of the etiology of Argyll-Robertson pupil, but is suggestive of the possible cause, this suggestion being further strengthened by two facts: (1) Although I found occasional axis cylinders of large size in oculomotor nerves supposedly normal (cases dead of cerebral hemorrhage), yet the difference between the normal nerve and the nerves from any one of the three cases cited is very great; (2) in an undoubted case of tabes with Argyll-Robertson pupil I found the same oculomotor condition as in my three cases, *i. e.*, swollen axis cylinders.

Ramak[6] says: "Degenerative Paralyses des äusseren Accessorius scheint bei Polyneuritis nicht beobachtet zu sein." I have not met in literature with trochlear nerve involvement nor with any obtunding of the sense of smell due to multiple neuritis. Starr[7] states that the eighth nerve has been involved in multiple neuritis, but he gives no instance.

With the above exceptions all the cranial nerves have been affected. The motor cranial nerves are much more vulnerable in multiple neuritis than the sensory.

In Case X the opthalmologist reported a high-grade neuroretinitis without hemorrhage in both eyes. In Case VIII, although there was no anesthesia in the distribution of the trifacial nerves, yet the points of exit of the supra-orbital and infra-orbital branches on each side were painful on pressure.

In Case I it is possible that there was some seventh nerve involvement, but this is far from certain.

The partial deafness which is so frequently seen in locomotor ataxia may be due to an involvement of the eighth nerve in a degenerative process similar to that affecting the oculomotor nerves in my three Cases (I, II, and III) of multiple neuritis and the one control case of locomotor ataxia examined.

It was von Strumpell[8] who first advanced the theory that the acceleration of the pulse in multiple neuritis was due to an involvement of the vagus. Later, Déjerine[9] confirmed von Strümpell's theory by finding in these cases a parenchymatous neuritis of the vagus histologically. But, as Remak[10] says, all cases of tachycardia in multiple neuritis cannot be ascribed to vagus involvement.

The following cases in my collection showed a rapidity of pulse which makes vagus lesion probable: VII, IX, XI, and XV.

In these cases the pulse varied between 110 and 140 per minute, with a temperature approximately normal. A number of the other cases had very rapid heart action, but it was either immediately before death or a high fever made it impossible for one to conclude that vagus involvement was the etiological factor.

In Case XI there was present a moderately high fever of an irregular type—though tending to be up in the evening and down in the morning. But the rapidity of the pulse did not seem to be influenced by this and was at times high when the temperature was subnormal. During the last four days of life the pulse frequence fell greatly, being usually between 58 and 64.

There is nothing which is not perfectly consonant with reason in supposing that a circulatory poison, such as is most probably present in all cases of multiple neuritis, be they caused by alcohol, lead, tuberculosis, diabetes, pregnancy, or the puerperium, if sufficiently active or if the tissues be sufficiently depressed in their resisting power, should be able to affect the central nervous system—brain or spinal cord—as readily as the peripheral nerves.

The axonal type of degeneration seen in the anterior cornual cells in cases of neuritis is not the action of the poison on the perikarion, but is, as Marinesco describes it, "réaction à distance." In like manner it is to be understood that where degenerative change has taken place in the posterior columns as in Cases IX and XIV, it is due to the fact that the process has extended far enough up to involve the cells of the posterior root ganglia, and the degeneration seen in the posterior columns still involves only sensory neurones of the first order. Degenerating axis cylinders with their medullary sheaths were easily traced, entering the cord via the posterior nerve roots in Cases X and XIV.

It is very seldom that the process is so severe that one is able to find severe degeneration of the emerging anterior roots, but in Case XIV not only was it possible to see that the anterior nerve roots were degenerated, but in the lumbar region I was able to trace by the Marchi method the separate strands of entering anterior root fibers from the periphery of the cord into the anterior horns of gray matter.

Spiller considers degenerative changes in the white matter of the spinal cord in multiple neuritis a rarity.

Ed. Flatau[11] reports a case of L. Jacobsohn's of a woman who had a carcinoma of the left mammary gland. There was total monoplegia involving the left upper limb, but at the histological examination of the left brachial plexus there was found no carcinomatous infiltration of the plexus, but simply an intense degeneration. There was no carcinomatous involvement of the spinal column or cord. On microscopic examination Burdach's column on the left was degenerated by the Marchi method in the region of the entering brachial roots, but on the right the posterior column was normal.

In Cases X, XII, XIII, and XVI swollen axis cylinders were found in goodly number in the crossed pyramidal tracts of the spinal cord. And in Case XIV there were found swollen axis cylinders not only in the crossed pyramidal tracts of the cord, but also in the motor pathway in the mid-olivary region of the medulla, and in the motor cortex the large ganglion cells were involved in a pronounced axonal degeneration.

This subject of central involvement is dealt with by Adolf Meyer,[12] who selects the term "central neuritis" to convey the idea. The term seems to me poor, not only because the concept of this central process as a neuritis is wrong, Meyer's elucidations (pages 105, 106, loc. cit.) notwithstanding, but because it might confuse where it was desired to differentiate between a neuritis confined to the extreme peripheral distribution and one of a more proximal type.

In order to say that a case of multiple neuritis has incontinence of the bladder and rectum because of disturbance of the nervous mechanism through neuritic involvement, it becomes necessary to exclude a number of factors frequently present. The incontinence must occur sufficiently long before death not to be classified as the incontinence of the moribund. There must be an absence of the profound asthenic condition sometimes noted in which involuntary evacuation of bladder and rectum is without pertinence. Also there must not exist a psychosis or suspension of consciousness. With these strictures the consensus of opinion has been that incontinence of the bladder and rectum is a rarity in multiple neuritis.

Von Leyden and Francotte have both reported cases of undoubted authenticity.

In my cases a true incontinence of bladder and rectum, not due to a moribund state, apathetic or abnormal mental condition, and apparently consequent to a neuritis process, was present in Cases III and XI. The histological examination of the cord was negative, with the exception of the axonal reaction in the anterior cornual cells, which is an effect of this neuritis and not a primary cord lesion. In Case III the incontinence came on seven days before death; in Case XI, six days elapsed before the end.

In Case II there was difficulty in starting the stream in micturition for three and a half months prior to death.

Cases V, VI, and IX presented incontinence of the bladder and rectum for some time before death, yet the psychosis renders it necessary to exclude them from consideration. For the same reason I exclude Case X from the list of true multiple neuritis incontinents, although his mental aberrations were not of a continuous nature and probably did not play a part in his bladder and rectal difficulty.

The subject of muscular spasm, or it might better be called motor irritation, is next in order for discussion. There has been much difference of opinion as to the etiology of these motor manifestations, chiefly because the cases presenting them, which have come to autopsy, are very few. Remak[13] has called attention to the rarity of fibrillary tremors, cramps, and clonic contractions of voluntary muscles in cases of neuritis, and Rossolimo[14] has reported two cases of pseudo tabes peripherica, in which there were choreiform movements, in one of the cases of all four extremities, in the other of the lower extremities. He calls these cases "L'amyotaxie," and the final summing up of his paper is: "Sous ce nom d'amyotaxie, on doit comprendre les contractions convulsives involontaires et de caractère réflexe qui accompagnent parfois l'ataxie et qui ont pour base aussi bien les affections des régions sensitives que celles des régions motrices du système nerveux et, le plus souvent, les névrites multiples."

In Case VII, access to which I have had through the kindness of Dr. Spiller, twitching of the muscles of the right lower limb began one month after the onset of his illness. This twitching was more than a fibrillary tremor, and looked like the jumping of entire muscles without producing the spontaneous movements of the limb spoken of by Remak and others. Very soon this muscular twitching gradually increased in extent and severity, and in about ten days from its onset had involved all four limbs and the two abdominal recti.

This case is very interesting because during its progress there was present a psychosis not unlike the Korsakoff syndrome. Now, most of the cases of motor manifestations have been noted in alcoholic multiple neuritis. The Korsakoff psychosis is also a condition due to alcoholic poisoning, yet in this case both of these symptoms—the muscular spasms and the psychosis—are present, and yet alcohol plays no part in the etiology of the multiple neuritis.

In Case VIII contractures of the lower limbs began to develop sufficient in severity to necessitate the use of splints. There was also tremor of the hands, and alcohol was the cause.

Although there are a number of things in Case VII which are in favor of a diagnosis of multiple neuritis, yet the exaggerated reflexes in the lower limbs and ankle clonus are certainly foreign to the conception of lower motor neurone disease. The increase in tendon reflexes in multiple neuritis is generally explained by accepting the theory that the neuritic process chiefly affects the sensory system, but only sufficiently to magnify, on account of the increased irritability, the afferent impulses, thereby causing an increased reflex.

Von Strumpel and Moebius[15] have reported two cases of increase of tendon reflexes in multiple neuritis.

Remak[13] says: "Es ist hervorzuheben, dass die soeben besprochene Steigerung der Sehnenphänomene bei Neuritis stets nur eine Ausnahme ist, und dass ein neuritischer

Process als solcher die Sehnenphänomene der nicht von demselben ergriffenen Nervenmuskelgebiete in der Regel unbeeinflusst lässt. But farther on he says (page 313, speaking of disseminated amyotrophic polyneuritis): "Die Sehnenphänomene sind in den atrophischen Gebieten geschwunden, konnen aber in anderen gesteigert sein."

The blue line on his gums, the abdominal pains, the absence of sensory involvement, the weakness of the extensor group with the preservation of the supinator longus in each upper limb, his employment in the chemical works—all of these points—supported by the negative finding in the spinal cord, make me classify this case as most probably one of multiple neuritis.

Remak[17] says that anomalies of the pupil, particularly difference in the size, are present in alcoholic multiple neuritis. He mentions the important work of Uhthoff,[18] who found, in 1000 cases of alcoholism, pupil anomalies in 6 per cent. Of these, 25 cases showed well-marked difference in size of the pupils, and in about an equal number very little reaction to light. In 10 cases, or 1 per cent. there was loss of puplillary reflex to light, but almost always was the reflex in accommodation and convergence preserved. In studying my cases, I find a difference in the size of pupils in Cases II, VI, IX, and XIII. Alcohol was a factor in 2 of these cases, VI and IX, but not a factor in Cases II and XIII.

Case XIV was reported by Drs. Spiller and Longcope,[19] Case XVII was reported by Drs. Spiller and Weisenburg.[20] Case X was reported by Dr. Spiller.[21] Cases IV and V have been reported by Dr. Mills and myself.[22]

In conclusion, I would say that Dr. Spiller has been prodigal in his generosity to me. The wealth of his laboratory has been unreservedly placed at my disposal. Any merit that might accrue to my work I gladly attribute to his great stimulation, which has meant so much to me and many others here in America.

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SACRAL TABES: A CASE WITH NECROPSY[1]

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Notwithstanding the fact that clinical cases of tabes, with preservation of the patellar tendon reflexes have been reported, the opportunity rarely arises to study this phenomenon pathologically. As far as I have observed, in a review of the literature two other cases, one by Achard and Levi, in the *Nouvelle Iconographie de la Salpêtrière,* Vol. XI, the other by T. Pineles, in *Obersteiner's Arbeiten,* 1896, have been reported with pathological findings, similar to those in this case. However, Redlich[2] and Pick[3] each have reported cases in which the sacral segments were chiefly involved, but these were cases of tabes dementia paralytica.

The patient, G. W., æt. 54, was admitted to the Philadelphia Hospital and was in the service of Dr. W. G. Spiller, through whose kindness the clinical history and pathological material were obtained.

The clinical history shows that one and one half years before admission the patient commenced to have sharp pains confined to his right thigh. Since his admission he has been confined to bed because of his ataxia. He has had incontinence of urine and feces during this period.

The family history and previous medical history are negative.

Examination shows an ill-nourished, markedly emaciated old man. Right eye shows a cataract, the cornea is ulcerated. Left pupil very myotic and reaction to light is questionable. Ocular movements are normal, associated movements are good. There is emaciation in the muscles of the shoulder girdle and upper limbs. The muscles are flaccid and soft, hypotonicity is distinct in the upper limbs. Power, as shown by the grip test, is fair on the right and slightly more on the left.

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[1] Read before the Philadelphia Neurological Society, November 27, 1908.

Triceps and biceps tendon reflexes are present on each side.

The ataxia as shown by finger-to-nose test is slight. The lower limbs present the same general emaciated condition of the muscles; power and resistance to passive motion are diminished. In heel-to-knee test, ataxia is present in both limbs. There is no tenderness over nerve trunks, and no fibrillary tremors. Achilles tendon reflex is absent on both sides. No Babinski or ankle clonus.

The patellar tendon reflex is obtained on each side, and is very prompt, especially in view of the marked wasting of the thigh muscles. While the leg is not thrown forward on the right side, and only slightly on the left, the contraction of the quadriceps is marked.

No mental disturbance is to be noted; he remembers past events fairly well.

Several months later the patellar tendon reflexes were examined again by Dr. Spiller and found to be of normal response.

At a later date, some difficulty was experienced in obtaining these reflexes, but a prompt response occurred on the left side, as shown by contraction of the quadriceps muscle, with very little movement of the leg. On the right side the reflex was not quite so prompt. At this time the eye examination showed the pupils contracted and reaction to light questionable.

Clinical Summary.—The hypotonicity and ataxia, the shooting pains of the lower limbs, the loss of Achilles tendon reflexes, the vesical and rectal symptoms, the myosis, make it clearly a case of tabes dorsalis. The prominent feature of the clinical history is the nearly normal condition of the patellar tendon reflexes with so pronounced tabetic symptoms.

Sections were examined from the following segments of the spinal cord: The eighth cervical, eighth thoracic, first, second, third and fourth lumbar. From the fifth lumbar the cord was cut into small pieces, as it was impossible to divide it according to the root segments. All the sections were stained with the Weigert, hemalum and Nissl stains.

From the last sacral segment to the fifth lumbar the posterior columns were markedly degenerated, especially on the right side, involving the whole of the root entrance or cornu radicular zone of Marie, which contains the medium and long fibers, forming the reflex collaterals and ascending fibers of the posterior columns. On the left side the root entrance zone was much less destroyed and many normal fibers entered the posterior horn. The pathological picture corresponded to the clinical findings of vesical and rectal disturbance, and loss of Achilles tendon reflex. In view of the preservation of some of the reflex collateral fibers on the left side one might have expected some evidence of the Achilles reflex on this side. However, we know that it is not necessary for all the fibers to be destroyed to cause the failure of the reflex. This is well proven in those cases of tabes with secondary hemiplegia in which a return of knee-jerk occurred.

Fourth Lumbar Segment.—At this level the root entrance zones are markedly degenerated, the sclerosis extends to the posterior horns. The posterior roots are very much degenerated, while the anterior roots are normal.

Third Lumbar Segment.—In this segment the sclerosis is wedge-shape, with its base at the periphery, the apex at the posterior commissure. This triangular area of degeneration is more marked on the right side, its extent being less than in the fourth lumbar. This is the highest level at which the posterior root fibers entering the cord are distinctly degenerated. The sclerosis of the posterior roots of this segment is slight and nearly equal on the two sides, though the right side is a little more degenerated. The anterior root fibers are normal. The pathologic findings, in this segment which shows the nearly complete preservation of the reflex collaterals, explain the clinical phenomenon of the preservation of the patellar tendon reflex.

Second Lumbar Segment.—The triangular area of sclerosis on the right side is smaller than in the preceding segment. A corresponding degree of change is noted on the left side, the area being less pronounced. The root entrance zone on the right side is considerably sclerosed along the periphery of the cord, although the fibers entering at this level seem to be intact. This degeneration is probably due to the loss of ascending fibers from lower segments. On the left side the root entrance zone is about normal. The posterior root fibers show some slight sclerosis. The anterior root fibers are normal.

First Lumbar Segment.—In this segment the root entrance zone on the left side is normal, the right root entrance zone shows very slight sclerosis. The triangular area on the left being faintly noted as similar to the right side. The left posterior root fibers are almost normal, the alteration is so slight as to be questionable, the right posterior root fibers show very slight degeneration. The anterior root fibers are normal. In this section as well as in the second and third lumbar segments, the root fibers were cut from the cord and many sections studied.

Eighth Thoracic Segment.—The column of Goll on the right side is much more degenerated than on the left. The columns of Burdach, root entrance zones, and cornu-commissural zones on each side are not involved. The anterior and posterior root fibers show no changes.

Eighth Cervical Segment.—This segment shows a similar picture as seen in the eighth thoracic. The column of Goll on the right side is much more degenerated than that on the left. The columns of Burdach are not involved. The same is true of the root entrance zone and cornu-commissural zone of each side.

With the hemalum and acid-fuchsin stain all the segments show the same picture. The pia arachnoid shows a slight thickening with slight round cell infiltration.

With the Nissl stain the cells of the anterior and posterior horns are found considerably pigmented.

Pathological Summary.—The degeneration of the posterior root zone was pronounced through all the sacral segments, and extended as high as the fourth lumbar segment. It was slight at the third lumbar segment, and in the middle thoracic and lower cervical segments only the columns of Goll were involved. The degeneration was throughout more pronounced on the right side and involved the posterior root zone from the fourth lumbar down on the right, whereas on the left side in the corresponding area many normal root fibers entered the posterior horns.

In this case preservation of the patellar tendon reflexes and the disappearance of the Achilles tendon reflexes are satisfactorily explained by the pathological findings. Ordinarily in tabes the lumbar segments are involved, and the typical symptoms arise, including loss of patellar reflex. In this case the sacral segments showed pronounced degeneration, the lower lumbar segments were involved, but the pathway for the reflex arc had not been destroyed. The slight involvement of a few of the fibers of the reflex collaterals of the right side corresponds to the finding of a slight diminution of the right patellar tendon reflex.

Achard and Levi, in 1897, reported a similar case, in which the diagnosis of tabes was made during life. The persistence of the reflexes was the chief feature. The patient had the Argyll-Robertson pupils, tabetic gait, and Romberg sign. The reflexes were examined at different times and showed no diminution or exaggeration. The microscopical examination showed, as in my case, extensive sclerosis in the sacral segments, especially in the inferior, while the upper sacral and all the lumbar segments showed a limited area of sclerosis. T. Pincles describes a case of sacral tabes in a patient aged 46 years, in which besides

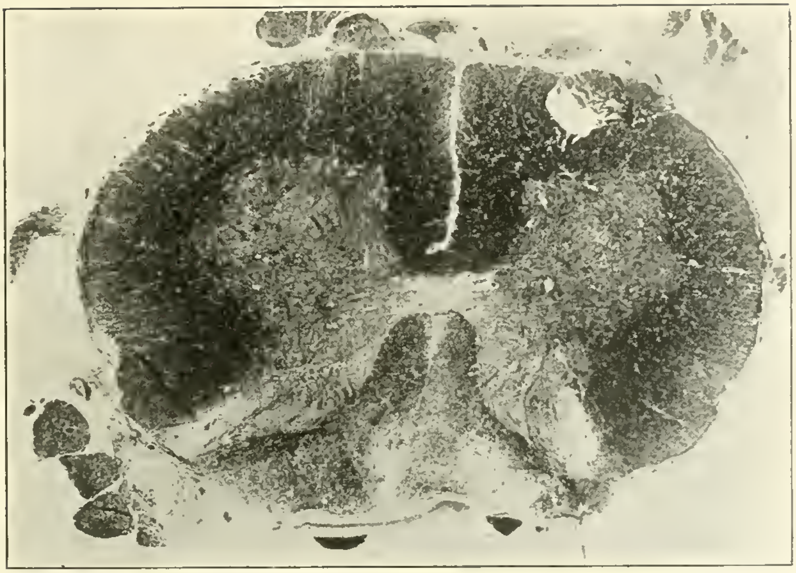

FIG. 1. Photograph of a section from the fourth lumbar segment. The hole in the anterior column indicates the right side.

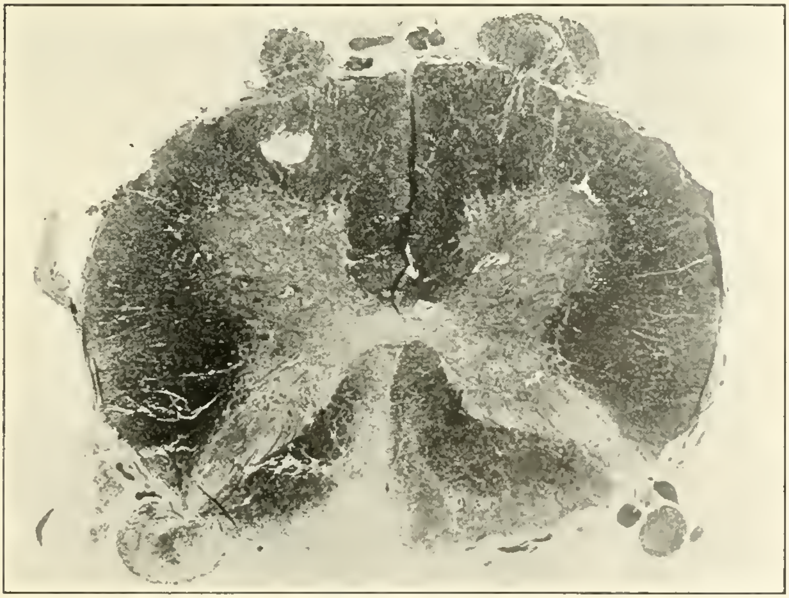

FIG. 2. Photograph of a section from the third lumbar segment. The hole in the anterior column indicates the right side.

lancinating pains and Argyll-Robertson pupils, the left patellar tendon reflex was lost, while the right patellar tendon reflex was obtainable only by reinforcement. There was no ataxia or Romberg. He speaks of it as a case of incipient tabes, in which the disease began in the lower spinal segments.

Redlich[2] describes in his monograph on tabes a case of tabes dementia paralytica in which the preservation of the patellar tendon reflexes was noted, together with loss of Achilles tendon reflex. The tabetic changes were found in the lower lumbar and sacral segments. In the upper sacral and lower lumbar segments the picture was that of an incipient process, while the lower sacral segments showed scarcely any changes. In my case an incipient process was not to be considered in as much as the degeneration throughout the sacral segments was so extensive.

In Pick's[3] case there was left facial paralysis, and the left patellar reflex was absent, while the right was only obtainable by Jendrassik's method. Sections showed intactness of the reflex arc in the lumbar segments.

Babinski and Nageotte, in 1905, reported their findings in a case, but according to the findings it was not one of the sacral type.

The unique process in their case I will discuss later; let us now return to the question of the patellar tendon reflex as an early symptom. What emphasis should be laid upon this symptom? Westphal in his first study of the knee-jerk said, "it is present when the posterior columns are involved only as far as the lower thoracic and upper lumbar segments, and also when the lateral pyramidal tracts are involved." Clinical cases have been reported by Hamilton, Erb, Theme, Berger and others, in which the patellar tendon reflexes were preserved. Berger found it preserved in 2.4 per cent. of 82 cases examined. Abadie found in 196 cases 13.5 per cent. with preservation of the patellar tendon reflexes. The studies of Jackson and Taylor, Goldflam, Mamlock, Dercum and others in cases of tabes with secondary hemiplegia and return of the patellar tendon reflex serve to show us the variation of this reflex.

To-day more stress is laid upon the disappearance of the

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[2] Redlich, E. "Die Pathologie der tabischen Hinterstrangserkrankung," 1897.

[3] Pick. "Anatomische Befund bei einseitigen Fehlen des Knie phaenomens." Arch. f. Psych., Bd. xx.

Achilles tendon reflex as an earlier symptom. Bregman quotes Babinski and Goldflam's view that the Achilles tendon reflex is more frequently and earlier altered than is the patellar reflex. The latest studies of collected cases of tabes seem to indicate the correctness of this statement. Kollarits, a Hungarian observer, found the Achilles tendon-jerk absent in 65 of 100 cases examined, while the patellar tendon reflexes were absent in 56, thus making 11 cases with absent Achilles tendon reflex as the only sign. Sarbo analyzed 92 cases and found the patellar tendon reflex abnormal in 87 per cent., the Achilles tendon reflex abnormal in 91 per cent. Bregman asserts from these, together with the sensory findings, that the sacral portion of the spinal cord is the earliest portion affected in tabes.

How shall we account pathologically for these variations in the patellar tendon reflex? We have seen that tabes may occur in which the patellar tendon reflex is preserved; again, we see cases in which the reflex disappears only to reappear. The view of most observers to-day is that the integrity of the patellar and Achilles tendon reflex depends upon two factors: first and chiefly, the preservation of the fibers forming the reflex arc, and secondly, upon a cortical influence whose pathway may be in the pyramidal tract, or in the cerebellar tract. If the reflex arc is entirely destroyed, then the patellar tendon reflex is entirely lost, and this is the picture described in most cases of tabes, pathologically the most pronounced degeneration is seen in the lumbar segments. If these segments are only partially destroyed then the patellar tendon reflex may or may not be present, or it may be absent and return. This phenomenon was seen in those hemiplegic tabetics, and depends upon the removal of cortical inhibition by degeneration of the pyramidal tracts.

In Pick's case where there had been return of the knee-jerks, the pathological findings explained the symptoms, in that only a portion of the reflex arc had been destroyed, and with the secondary advent of the destruction of the pyramidal tract the removal of inhibition occurred upon the few fibers of the reflex arc left intact. In Achard's case, in Redlich's case and in my case, the preservation of the reflex was explained by the involvement only of the sacral and lower lumbar portions of the cord.

Babinski and Nageotte[4] found an inverse systematization in their case. A young man, 25 years of age, had tabetic symptoms, with preservation of the patellar tendon reflex. On examination of the sections the degeneration involved the long fibers of the posterior roots, or in other words, the postero-external zone. The oval field of Flechsig and the columns of Goll presented lesions of the same intensity while the short or medium systems of fibers, those usually attacked first in this disease, escaped. This is contrary to the usual onset of this disease, consequently they speak of it as an inverse systematization in tabes. No mention, however, is made of the sacral segments. The important observation to be deduced from my case, and those cited is that, clinically, tabes should not be overlooked in those cases in which the patellar tendon reflex is preserved. The early disappearance of the tendo-Achillis reflex is attaining greater significance, and as indicated above, among some observers, as Babinski and Bregman, is of greater value as an early sign.

Besides this case also shows the polymorphic nature of this disease, and to-day instead of only the lumbar type, the cervical type, the optic type, and paralytic type, we have pathologically and clinically the sacral type.

I wish to take this opportunity to thank Dr. Spiller for his kind assistance in the examination of the pathological sections.

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[4] Babinski and Nageotte. "Note sur un cas de tabes a systematization exceptionelle." Soc. de Biol. de Paris, 1905.

CAUSE OF CONTRACTURES AND SPASTICITY IN CASES SHOWING NO DEMONSTRABLE LESION OF THE PYRAMIDAL TRACTS WITH A PATHOLOGICAL REPORT OF THREE CASES[1]

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The three cases to be reported in this paper are examples of at least unusual causes of contractures and spasticity evidently of central origin clinically, in which, however, at autopsy there was no demonstrable evidence of any disease in the pyramidal tracts in two cases, and slight, if any, in the third.

In the first case, clinically, the patient presented the symptoms of a bilateral hemiplegia, with marked contractures of arms and legs. In the second case, one of apraxia (26), previously reported, there was general rigidity of the limbs, and spastic contractures of the left arm and, to a less extent, the left leg, were present.

In the third (27) case, also already reported, that was spastic paresis of the legs, dating from childhood, in a man who died at the age of 72. The discussion of the cause of the contractures in the first two cases will occupy our attention first.

Contractures may result, as v. Monakow (15) described, from (1) irritation of the sensory portion of the reflex arc, such as inflammatory processes in and around the joints, nutritive disturbances of the muscles, injury or inflammation of the peripheral nerves; (2) toxic and mechanical irritation around the motor nuclei, which does not injure them materially; (3) irritation or interruption of the cerebral connections, such as inflammatory foci, meningitic processes, circulatory disturbances and the like,

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[1] Read by title at the thirty-fifth annual meeting of the American Neurological Association, May 27–29, 1909.

as well as similar causes operating upon the cerebellar connections and tegmental tracts (Haubenbahnen).

There are also contractures that are associated with hydrocephalus, the cause of which is still open to discussion; and contractures in psychopathic and functional nervous conditions, and in diseases of the spinal cord. It is the contractures that are caused by lesions of the cerebro-spinal system which will be discussed here.

While the causes of contractures in cases in which the pyramidal tracts have been found intact have been the subject of some discussion in the literature, there are very few cases of this sort on record. This phase of the question was discussed in the original paper, which contained the report of my third case.

The references in the literature to the cause, or causes, of contractures concern mainly those occurring in connection with hemiplegia, and in which there is a lesion somewhere in the motor tracts.

It would lead me too far afield to enter into an exhaustive discussion of the various theories advanced to explain the origin of hemiplegic contractures and spasticity; at the same time it is impossible to avoid a review of these theories in order to determine what relation they bear to the etiology of the contractures in the cases to be cited in this paper, in which the pathology differs so much from that commonly found in cases of hemiplegic contractures.

The early theories that hemiplegic contractures were due to exaggerated associated movements (Hitzig (10, 11)), or to secondary degeneration of the pyramidal tracts (Bouchard (1)), or the products of the destruction of the degenerated fibers, causing irritation of the anterior horns, thus giving rise to contractures (Charcot (2)), are no longer tenable.

The influence of the optic thalamus and subcortical centers upon the development of these contractures has been invoked by several authorities, namely, von Monakow (16), Rothmann (24), Förster (4) and Grasset (5).

Von Monakow looks upon the late hemiplegic contractures as the result of a disturbance of the kinetic equilibrium, and of the nature of an incomplete motor compensatory action arising from an extensive destruction of the highly developed central movement-components, particularly the primary fibers. According to his views the compensatory action is supplied in part by the gray substance of the spinal cord, and in part by the philogenetic subcortical centers. Lesions causing hemiplegic contractures usually do not materially destroy the sensory tracts, and the preservation of these, and the destruction of the motor tracts establish a disturbance of the equilibrium in the delivery of the motor impulses. Centripetal stimuli flow in excess to the motor centers, and there is a false distribution of the sensory stimuli. The subcortical centers become chronically irritated, and this gives rise to the delivery of simple kinetic impulses, causing contractures which correspond in degree to the size of the cross diameter of the muscle.

Rothmann believed that the hemiplegic contracture, as well as the return of voluntary movement in the paralyzed muscles, are the result of an independent motor function of the optic thalamus and corpora quadrigemina, which gradually develops, rather than a return of the function of the pyramidal tracts, or a compensatory function of the cortex of the other side. Lewandowski denied that this was proved.

Förster claimed that the cause of the contractures is a subcortical fixation reflex situated in the subcortical centers and which operates in its full strength when these centers are severed from the inhibiting influence of the cortex. According to Förster these centers do not act at once after this influence is cut off, but it is only after a certain time that this function of the subcortical centers manifests itself. This is not the only cause, but is associated with an increase in the tendon and periosteal reflexes and skin reflex, as well as the production of associated movements. According to him the character of the contracture depends upon the factor by which the insertion points of the muscle are approximated, and that there develops a progressive involuntary contraction, in which condition of shortening of the muscle there is an inclination of the muscle to become fixed. In other words, the character of the contracture depends upon the tendency of the muscles to accommodate themselves to an approximation of their insertion points, and to be fixed in this position. He believes that the contracture is a subcortical fixation reflex.

Mann (17) criticizes Förster's position in this discussion, and states that he was unable to cause a change in the contracture by passive change in the position, and also states that he has observed a flexor contracture from passive position change to an extensor contracture when there was a return of active movement in the latter muscles. Mann also objects to the position taken by Rothmann, on the ground that the restitution of motion is a return of voluntary movement, not an automatic one, and believes that the hemiplegic contracture and the restored movement originate in the medium of the same tracts; also that paralysis and contractures may result from injuries beneath the subcortical centers.

In a recent paper Mann no longer adheres to his early views that there are excitomotor and inhibitory fibers for each muscle, *i. e.*, that excitomotor fibers for certain groups of muscles are identical with, or run along with the inhibitory fibers for their antagonists. His present view is that in purposeful movements there flow out, besides innervation impulses to certain muscle groups, denervation impulses for their antagonists.

As recently expressed his theory for the development of these contractures is that they, as well as typical residuary hemiplegic palsy, result from a *partial* interference with the conduction along the central motor fibers. These impulses lose their harmony to a certain degree, by which some muscles suffer in their capacity to contract, and others to relax, developing a disharmony which expresses itself in a lack of tone on the one hand and an excess on the other. They are due, in other words, to an innervation of the agonists and a denervation of the antagonists. Mann sees a constant relation between the restitution of power and the development of contractures and claims that the latter are the result of *partial* destruction of the pyramidal tracts.

Lazarus (12) believes that the interference in the conduction of the pyramidal tracts is associated with a cutting off of the inhibitory influence from the cortex, resulting in a hypertonia of both agonistic and antagonistic muscles. According to him the normal muscle tone represents a reflex act dependent upon the integrity of the spinal and spino-cerebral reflex arcs at the same time. When the spino-cerebral arc is destroyed, and the spino-reflex arc is intact there results a loss (Ausfall) of the inhibitory impulses from the cerebrum, which is expressed clinically as hypertonia and increased reflexes. Paresis, he states, always is associated with hypertonia, and they are both the result of disturbance of conduction in the pyramidal tracts, and he criticizes Mann's idea of a partial loss of conduction in the pyramidal tracts as a cause of the hemiplegic contractures. The dissociated palsy is not, according to Lazarus, the result of a removal of the relaxing impulses for the agonistics, but is a dissociated muscle arrangement. The palsy in the muscles, Lazarus holds, is relative to the normal superiority of the muscles, contracture developing in that muscle which is relatively superior in strength and tone; and, according to him, the more complete the palsy the more extensive the contracture. The characteristic hemiplegic contracture corresponds to the physiological superiority of certain agonistic functions over their antagonistics.

Mann (18), however, in response to Lazarus's criticism, states that total destruction of the cord does not cause the greatest degree of contracture, and that there are cases in which total destruction of the pyramidal tracts has not been associated with contractures at all, and he believes that the contractures are more conspicuous in those cases in which certain muscle groups are palsied, while their antagonistics are preserved, and therefore, when the motor conduction is partially intact.

But Lazarus (13) raises the objection that the typical extension of the paralysis of internal capsule origin to the entire half of the body is an expression of total destruction of the pyramidal tracts, and that it is not compatible that in this form of paralysis the flexors of the arm remain intact, and the extensor fibers are destroyed. He further calls attention to the fact that an analysis of the paralysis in typical hemiplegia shows in all the muscles a loss of power; never are the antagonistics entirely exempt. They are not only hypertonic, but paretic, as is seen in efforts to overcome the contractures, and he does not believe with Mann that the reflexes are increased only in the agonistics.

An effort has been made by some authorities to connect hemiplegic contractures with disturbances of inhibition, which is exercised by the cortex through the pyramidal tracts upon the spinal cord.

According to Marie (20, 21) the cells in the anterior horns of the spinal cord are normally in a permanent state of activity—"une machine toujours sous pression,"—and the central neurone has an inhibitory action upon this activity. When communication between the cortex and the cord is interrupted the inhibitory influence is removed, contractures resulting in consequence.

Parhon and Goldstein (23), who accept Marie's theory, believe that the excitomotor fibers for one muscle group functionate at the same time as the inhibitory fibers of the antagonistic muscles. In hemiplegic contractures the excitomotor fibers for the contractured muscles are conserved, and the inhibitory fibers are implicated; while the contrary exists for the antagonistic muscles. These authors oppose the theory advanced by Grasset (5), who places an automatic center for muscle tone in the pons. Grasset believes that lesions of the spinal portions of the pyramidal tracts cause contracture by suppressing the inhibitory action of the tone which arises in part from the pons, and in part from the spinal cells.

According to Lazarus the removal of the inhibitory influence of the cortex is the cause of the hypertonia of the muscles.

Rothmann (25), however, does not believe that the disturbances of active inhibltion and its conducting fibers is to be considered in the explanation of contractures, while Lewandowski (14) believes that contractures are not caused by the removal of inhibition, but are due to persistent inhibition of the antagonistic muscles. The effect of position in causing contractures is not simply reflex, Lewandowski claims, but is also in part the result of certain volitional innervations.

He further states that if there is a complete break in the motor conduction there would be complete paralysis, and if the contractures were due to inhibition thus removed, all the muscles would be contractured; but this is contrary to the facts, for in complete palsy, he states, there are no contractures.

Van Gehuchten (7) holds that there are two sets of fibers connecting the cortex with the cord, (1) the direct cortico-spinal tracts in the pyramidal tracts, and (2) the indirect cortico-ponto-cerebellar spinal tracts. The latter are composed of two parts: (*a*) a cortico-ponto-cerebellar portion, and (*b*) the cerebro-spinal portion which connects the cerebellum with the cord through the inferior cerebellar peduncles. The direct fibers have an inhibitory action, and the indirect fibers are excitomotor. Lesions which injure the direct, or inhibitory tracts permit the predominance of the action of the excitomotor fibers, with the result of inducing contractures. These two tracts are associated with each other in the pyramidal tracts in their cerebral course, where lesions cut off all influence of the cortex from the cord, and cause a flaccid paralysis. In the spinal cord, however, these tracts are independent of the pyramidal tract, and lesions of the direct connections (cerebro-spinal tracts) or inhibitory fibers allow the predominance of the indirect excitomotor fibers, resulting in the causation of contractures. The protagonists receive a greater amount of innervation than the antagonists, in a word, and thus give rise to the contractures.

Gerest (6) raises an objection to this theory that in lesions of the cortical centers, destroying the cortical neurones, contractures may be absent, and that the late appearance of contractures is against van Gehuchten's view. He also raises the objection to van Gehuchten's theory that contractures occur in muscles in which the paralysis is less intense, but this is not true in peripheral neuritis. But van Gehuchten believes that there are always some persistent motor cells, in answer to Gerest's first objection, and that the late appearance of the contractures is due to the removal of the compression of healthy fibers, which causes the early flaccid paralysis; and, finally (in reply to the third objection), that in peripheral neuritis complete paralysis of muscular groups is not always observed, as in post-hemiplegic contractures.

Mann states, in objection to van Gehuchten's theory, that the contractures are an expression of a predominating innervation of the functionally preserved muscles over the palsied muscles; that in peripheral paralysis one would expect greater contractures than in hemiplegia; but the reverse is true. And also certain cases of high-grade contracture are found where the palsy is slight, and the reverse in hemiplegia. Moreover, peripheral contractures are different from hemiplegic contractures, being constant in the former and variable in the latter. And he goes further and states that in the development of the contractures there is sometimes a certain degree of hypertonia, where the voluntary motion has not returned, or only slightly so.

Van Gehuchten (9) criticizes the position held by Mya and Levi, who believe that the muscles remain inert after being cut off from their excitomotor centers, and that they then recuperate insensibly an independent function resulting in hypertonia, that is, contractures and increased reflexes. Van Gehuchten, however, states that when the palsy is not a flaccid one there is not entire interruption of the fibers, as Mya and Levi believe, and that moreover, after complete section of the cord, the complete flaccid palsy which results is not followed by contractures.

Another element in the causation of contractures is, according to von Monakow, the larger volume of the muscles, that is the static preponderance on one muscle group over another. In this connection Lazarus also calls attention to the element of superiority of certain agonistic muscles over their antagonistics.

According to Rothmann, van Gehuchten, Mann and Crocq (3), the contractures are associated with incomplete interruption of the motor conduction fibers. But von Monakow has seen hemiplegic contractures when there was complete resorption of both, or one pyramidal tract, and Lazarus believes that contractures depend upon total destruction of the pyramidal tracts.

Van Gehuchten believes that the contractures are associated with the return of power without which no contractures develop. Mann agrees with this view, and states that the hypertonia is located in the antagonistic muscles which preserve a certain degree of motion.

In all these theories the contractures are related to the interruption of the action of the motor cortex, either partial, as assumed by van Gehuchten (8), Mann (19) and others, or complete, as claimed by Lazarus.

In other words, it is assumed that all the causes of hemiplegic contractures are related in some way to implication of the motor tracts, or, in certain cases of infantile cerebral paralysis, to an agenesis of the pyramidal tracts.

In the first case to be reported here no lesion of the pyramidal tracts of the cord, medulla, or pons, or in the motor portion of the internal capsule could be found.

This case was that of a man aged 67, who was examined only a short time before death, and who at that time appeared to have double hemiplegia with contractures of the arms and legs. The forearms were partially flexed on the arms and the hands were held in a position of flexor contracture. The thighs were flexed on the trunk and both knees were held well over toward the left of the trunk. The legs were flexed on the thighs, the feet extended. There were also contractures of the neck muscles, the head being flexed markedly upon the trunk. Efforts to overcome these contractures were met with considerable resistance, especially in the shoulder-joints.

Sensation was apparently preserved to the sharp points of the esthesiometer.

The knee jerks could not be elicited, probably due to the contractures. The Babinski phenomenon was present on both sides. Only slight voluntary motion was present in the arms and legs. There was general wasting, but no local atrophy.

The family and previous history was unimportant. The first symptoms dated 31 months before death—suddenly. Contractures began to develop in about 6 months. There was no apparent disorder of vision or speech. This much of the history of the onset of the disease was obtained from his son, who was unable to give any further data. The mental condition precluded obtaining any information whatever from the patient.

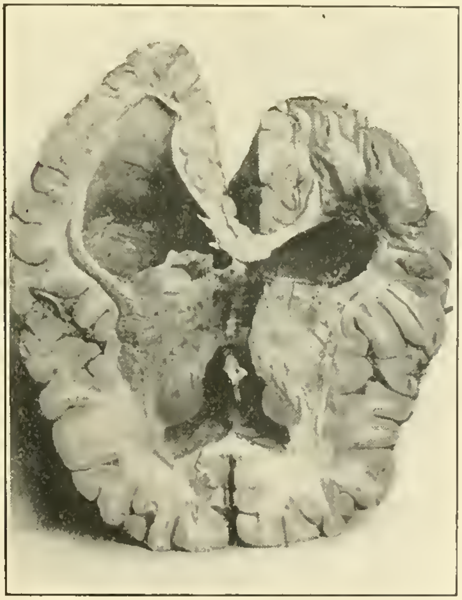

FIG. 1. Case 1. Hemorrhage into the wall of the posterior horn of the lateral ventricle.

Pathologically there was a hemorrhage in the outer and posterior wall of the posterior horn of the right ventricle which destroyed the fasciculus longitudinalis inferioris, the optic radiations of Gratiolet, and the tapetum in part. There was also a small area of hemorrhagic softening situated about 3 cm. below the superior border of the right hemisphere at about its middle portion, cutting the fibers of the corona radiata, and extending in a vertical direction for about half a centimeter. There was also a small hemorrhage in the left optic thalamus.

Serial sections were made of the brain and also of the pons, medulla, and various parts of the spinal cord.

Secondary degeneration was found in the occipital lobe, in the fasciculus longitudinalis inferioris, optic radiations of Gratiolet, and the tapetum. The white matter of the cortex going to the calcarine region stained well. Secondary degeneration of the white matter could also be traced from the small hemorrhage in the right corona radiata, in a downward direction, to the posterior segment of the internal capsule. This was very slight, not extensive, and could not be traced as far as the foot of the peduncle. Degeneration of the occipito-frontalis fibers was also noted. Nowhere, however, in the entire motor tract could any degeneration be demonstrated.

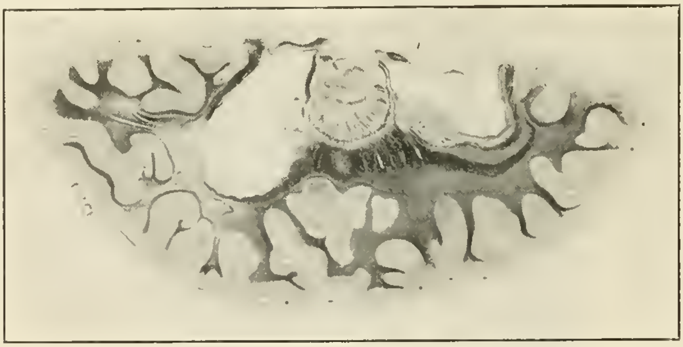

FIG. 2. Case I. Degeneration of the occipito-frontalis bundle and internal capsule.

The second case, already reported before this society last year, was one of apraxia occurring in a blind man of 55, who "was totally unable to designate the position of the limbs; could not locate touch anywhere; could not recognize objects by the sense of touch; and his touch and temperature senses were imperfect in the left hand. The left hand, though capable of some reflex acts, could not be moved voluntarily. The left hand was apraxic, and apraxic phenomena were present in chewing and walking."

"The left hand was held in a slightly contracted position, that is, partial flexion of the forearm upon the arm, and semiflexion of the fingers. This could be passively overcome almost entirely, but there developed at the same time a tremor of both arms, especially the left. There was also a slight but distinct rigidity of both legs and the right arm, but to a less extent than in the left arm.

"The autopsy revealed the presence of degeneration of the white matter of the right occipital and parietal regions on the convexity, and the posterior portion of the temporal lobe, the calcarine region remaining intact. The fasciculus longitudinalis inferioris and the optic radiations were degenerated on the right, and probably, though less markedly, on the left. On the left side there was degeneration in the occipital and temporal regions to a much less degree, leaving the median surface intact. The corpus callosum in its posterior portion was degenerated. Elsewhere the brain was apparently normal." Nowhere was there any degeneration of the motor tracts in the brain or cord.

The third case was reported before a recent meeting of the Philadelphia Neurological Society, and was that of a man who, at the age of 71, presented symptoms of spastic paraplegia dating from early childhood, and who, at autopsy, presented no gross lesions excepting an extensive effusion of blood in the pia arachnoid, extending over both hemispheres and the base, and evidently of recent origin. The pia of the cortex showed slight cellular infiltration, the blood vessels were somewhat thickened, and the cellular elements in their walls were somewhat increased. The cells of the cortex revealed no abnormalities, although no Betz cells were found.

Sections from the pons, medulla and spinal cord, by the Weigert method, showed no signs of degeneration whatever. Stained by hematoxylin and acid fuchsin the crossed pyramidal tracts on both sides took the stain slightly more intensely than the remaining white matter.

A further study of these fibers in the crossed pyramidal tracts showed that a number of them were very small and closely packed together, and that large, well-formed fibers were rare. There was no evidence of secondary degeneration of the pyramidal tracts. There was slight increase in the connective tissue in this region, but nowhere did it present the appearance seen in spinal cords in which the pyramidal tracts were the seat of secondary degeneration. The fineness of the fibers was very evident in comparison with the larger fibers in the cerebellar tracts.

In the first case, then, presenting shortly before death the appearance of a double hemiplegia with contractures of the arms, legs and neck muscles, there was pathologically, a hemorrhage in the right side of the brain involving the outer and posterior wall of the right lateral ventricle, which cut the optic radiations of Gratiolet, the inferior longitudinal bundle, and the tapetum in part, leaving intact, however, the gray matter.

There was also a small area of softening situated about 3 cm. below the superior border of the right hemisphere, at about its middle portion, cutting the fibers of the corona radiata, and extending in a vertical direction for about half a centimeter. This caused some degeneration of fibers in a downward direction, which could be traced into the posterior limb of the internal capsule for a short distance.

There was also a hemorrhage in the left optic thalamus.

As a result of the first lesion there was a degeneration of the white matter of the occipital lobe, and a degeneration of the occipito-frontal fibers.

In the presence of such pathological findings it is difficult to explain the clinical symptoms presented by this case. With practically intact motor neurones in the spinal cord, medulla and pons and cerèbrum, with lesions only in the sensory areas of the brain, we find nothing in common with the various theories cited above to explain the cause of hemiplegic contractures.

A possible, though not entirely satisfactory or convincing explanation may be found in the fact that the mechanism of motor-complexes requires the co-operation of the sensory centers. If the sensory centers are destroyed in certain localities—for example, in the occipital lobe—the sensory optic element is cut out of the motor complex, and there results a condition called psychic paralysis, in which the muscles lose their power of volitional contraction, except in reflex acts. The inaction of the limb involved affords an opportunity for certain mechanical forces to operate. Contractures would occur in the implicated members as a result, perhaps, of the predominance of one muscle group over another, in the way described by Lazarus and von Monakow, and, therefore, as a result of a cause independent of increased muscle tone.

This is only a possible explanation of the contractures in my cases, and a more plausible one for the case of apraxia, in which the contractures were unilateral, involving the arm suffering from psychic paralysis, than in the first case of bilateral contractures, with unilateral lesion commonly found in psychic paralysis.

Causes arising from dermatogenous, osteogenous, tendogenous, and arthrogenous sources may be dismissed without discussion. The contractures in these cases were evidently of central origin.

I do not believe that the small hemorrhage in the right corona radiata in the first case was sufficient to cause a double hemiplegia with bilateral contractures, as it is doubtful whether the motor fibers were implicated at all, and if so, the degeneration involved only some of the fibers of the leg on one side, as far as can be judged from a study of the lesion in the posterior limb of the internal capsule.

The effect of the lesion in the left optic thalamus is doubtful in the first case. It is known that disturbances of the optic thalamus result in the increase of muscle tone on the opposite side of the body (Muratow (22)), and that irritation of the optic thalamus with intact cerebellum produces tonic spasms.

If a lesion of the optic thalamus is an element in the cause of the contractures in my case it would only explain those on one side, and as the contractures were bilateral, it is therefore inadequate, in my opinion, to satisfactorily account for the conditions described.

It might be argued by some that the contractures in the first two of my cases were due to the internal hydrocephalus, but the explanation usually given for the cause of paralysis and contractures in hydrocephalus, *i. e.*, that they are the result of pressure upon the motor tracts, is not entirely satisfactory. I cannot imagine that any pressure upon the motor tracts, sufficient to cause an absolute paralysis and marked contractures, could exist for a long time without causing some degeneration of these tracts, and if the pressure is not sufficient to cause degeneration of these tracts, I do not believe it could cause a persistent total paralysis. Moreover, it is inconceivable that the pressure should affect the motor tracts exclusively, leaving the many other tracts of the brain intact.

Before one can accept the view that contractures in internal hydrocephalus are due to pressure, there must be a number of reports of cases of this sort in which the brain has been cut in serial sections in order to determine whether or not there has been any actual implication of the motor tracts in the brain.

The pathological conditions described in the third case represented, in my opinion, possibly a hindered, or poorly developed crossed pyramidal tract, or partial agenesis, and formed the basis of a possible explanation of the clinical manifestations.

In my previous report of this case I expressed the theory that the poorly developed pyramidal tract, consisting of fine fibers and perhaps also a diminution in the number of fibers, resulted in a lowering of the conductility of the fibers for the motor impulses from the cortex to the spinal cord. The presence of fibers in the pyramidal tracts I believe presupposed the intactness of cortical cells, or at least, cells somewhere in the cerebrum, and the character of the fibers and cells must have some bearing upon the development of the spasticity which this patient presented.

It seemed to me that when fibers of small caliber are called upon to conduct vigorous motor impulses we would meet with the same conditions as when currents of high potentiality are forced through conductors of small caliber, *i. e.,* increased resistance and imperfect conduction. It is conceivable that the increased resistance offered by the small fibers to the motor impulses could prevent the impulses from reaching the cells in the spinal cord *in toto,* thereby cutting off, partially, at least, some of the inhibitory influence of the cortical centers, contractures arising as a result of the action of the cells of the anterior horns of the spinal cord.

The first case, in my opinion, illustrates—though in a way not yet clear—that contractures may develop as a result of the implication of the sensory tracts independently of the motor tracts.

Lewandowski has already called attention to the influence of the sensory portion of the nervous system upon the causation of contractures, citing the fact that in tabes posthemiplegic contractures are practically unknown, and Sherrington's experience, in which, after cutting the posterior roots, the muscles relaxed more quickly than normal after cortical irritation.

Exactly how destruction of sensory intracerebral tracts causes these contractures is conjectural, but it is clear that they exercise a distinct influence upon their development.

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INDURATIVE HEADACHE

(SCHWIELENKOPFSCHMERZ) WITH REPORT OF THREE CASES *

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Indurative headache is of organic origin, chronic and usually paroxysmal, occurring in various parts of the head, and due to infiltrations or thickenings at various points, chiefly in the muscles of the head and neck. From the so-called cases cephalalgia, cephalodynia, migraine, hemicrania, cranial neuralgia and neuritis, many can be separated as belonging to this class.

A search made through works of neurology and monographs on headache by American physicians found little or no mention of headache due to these indurations. The subject seems neglected, except for the statement that headache is sometimes rheumatic, lithemic, or gouty in origin. The condition is ably treated, however, by Professor Edinger[1] and by Professor Norström,[2] of Stockholm, who spent some time in New York and while there wrote on this subject. The significance of this condition has been recognized for years by physicians abroad, notably by the Swedes and Germans.[3] In writing on headache due to organic disease, Edinger makes the astonishing statement that, though indura-

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[1] Edinger: In Diseases of the Nervous System, translated from Die Deutsche Klinik, Appleton & Co., New York, 1908.

[2] Norström: Der chronische Kopfschmerz und seine Behandlung mit Massage. Leipsic, 1903; Le migraine; traitement par le massage, Paris, 1904, Baillière & Fils; New York Med. Jour., 1905, lxxxii.

[3] Reidel: Der Kopfschmerz und seine physikalische Behandlung. Berl. klin. Wchnschr., 1907, xliv, 338; Kleen: Handbook of Massage, P. Blakiston's Son & Co., Philadelphia, 1892.

tive headache is almost unknown, it is probably the most frequent form of this disease.

CAUSE

In studying the cause of this condition we find that at the insertions or within the bodies of the muscles of the head and neck there appears a thickening which comes and goes at first and later becomes chronic, and which gives rise to stimulation or irritation of the sensory nerve fibers, thereby causing pain. Given, then, this chronic condition, a paroxysm may be brought on by emotional disturbance, physical or mental fatigue, sudden exposure to cold, as a chilling draft, insufficient drying after washing the hair, a stay in badly ventilated places, as the theater, by the approach of damp and chilly weather or storms. As Dr. Mitchell[4] wrote, "It is only required to come within the outer limits of storm conditions. The sky may be cloudless and the rain or cloud areas two or three hundred miles away and yet the sufferer be within the area of barometric depression. It was not needed that there be rain." The condition varies with seasons, being least common in summer and most frequent in fall and spring.

It is not the purpose of this paper to enter into the consideration of the pathologic chemistry of these swellings, for after much investigation the chemistry of gout, lithemia, uricacidemia and allied conditions is still far from being satisfactorily defined. These infiltrations or thickenings actually exist and give rise to a chronic myositis at the points of their lodgment. They are found present in three stages: 1. A swelling. This is of a soft, yielding consistency and is frequently present in the bodies of the muscles, where a puffiness is often noticed. 2. A slightly elastic resistance offered to the touch as though some organization had taken place. 3. Induration. There is an absence of elasticity and organization has advanced to the stage at which a substance of cartilaginous consistency presents itself to the examiner. The older these thickenings are the firmer they have become and the more resistance they offer to treatment.

These indurations appear again and again for some time before becoming chronic, and they are by no means confined to the head and neck. Other haunts of this insidious invader are the points where the gracilis, sartorius and semitendinosus muscles cross the inner side of the knee, at the inner and outer aspects of the ankle, at the insertions of the pectoralis major and latissimus dorsi into the humerus, the muscles of the abdominal wall where there are occasionally found little nodules which feel like small grains of shot, and which give rise to sharp pain on slight movement. In one obscure case of this kind the patient was suspected of having appendicitis. So-called cases of sciatica have ceased after the removal of these infiltrations around the gluteal and sacral regions. Deafness has been produced by the pressure of these swellings on the Eustachian tubes, and hearing has been restored after appropriate treatment. By far the most frequent site of these accumulations is in the muscles of the head and neck, and I believe that this is due to the fact that this is a much exposed part

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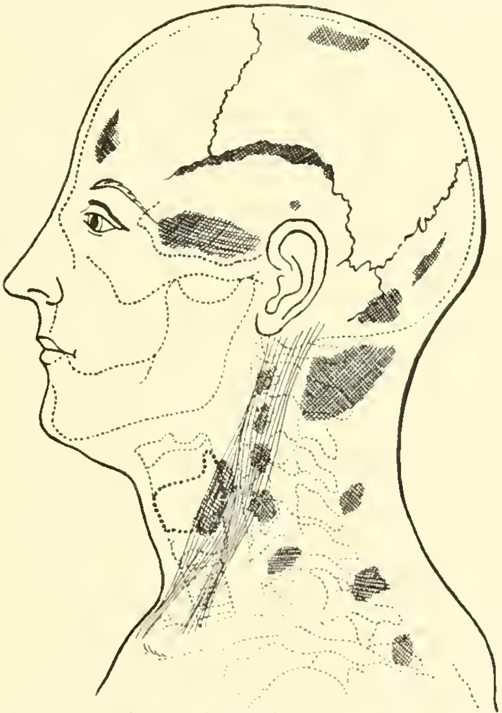
[4] Mitchell: Relation of Neuralgic Headache to Storms, Tr. Coll. Phys. Philadelphia, 1903, xxv, 71.



The most common sites of indurations. (Drawing modified from Edinger).

of the body. These indurations are probably the chief cause of the so-called rheumatic throat, but I do not believe any of these conditions are due to gout, for a paroxysm of gout, such as frequently involves the metatarsophalangeal articulation of the great toe, gives rise to marked redness and fever. An attack of lumbago does, however, present a strikingly similar picture, inasmuch as it frequently follows exposure to dampness or cold. After a number of acute attacks it may become chronic, offering considerable resistance to palpation. and it may give rise to a dull ache or sharp pain. It is relieved by local pressure. There is no redness and fever present, and it will yield to the same treatment employed in the removal of these indurations.

SYMPTOMS

The symptoms are of two groups—those of the chronic condition and those of the paroxysm. In the development of these pains, they are at first slight and infrequent; gradually they occur more often, are of greater severity and of longer duration. Sometimes they are of the nature of a dull ache and almost constant; again they may be sharp in character. Local pressure often affords relief, but if an actual neuritis is present the suffering will be intensified by pressure. These patients are very susceptible to colds, subject to depression of spirits and mental torpor and frequently there are associated symptoms in other parts of the body, some of which are gastrointestinal disturbance, toxemic in character, painful spasms of the calf muscles, an occasional attack of nephritic colic, myalgia in many different parts of the body, and the oral cavity may give manifestations in the form of hypersensitiveness of the teeth and pyorrhea alveolaris. It is probable that all these local conditions are but different manifestations of the same general disturbance of nutrition. Many of these cases are neurasthenic and it seems likely that an excess of uric acid or an allied substance is concerned in the production of this condition. Before an attack of headache the enlargements become swollen and sensitive; the pain which develops a little later may be mild or agonizing. It occurs in various parts of the head, depending on the location of the enlargements; it is usually over the site of these areas, but there may be radiation of the pain. Fever and redness are absent.

All cases of headache should be examined by forcible flexions and twistings of the neck, which may bring out intense pain at the insertions or along the bodies of the neck muscles. The exploration may be made advantageously during a paroxysm, for at such a time the swellings are more apparent and their sensitiveness greater, so that points which might otherwise be overlooked are then easily detected. The head and neck should be thoroughly palpated in the following manner:

Moderately firm pressure should be employed over the entire scalp, where thickenings, irregularities, indurations and nodules may be found. These parts may be only slightly sensitive or they may be exceedingly so. The same condition is then sought at the insertions of the trapezius, scaleni, splenii and sternomastoid muscles. Hypersensitive points are often found around the base of the skull from one mastoid process to the other, on the spinous processes of the cervical vertebræ, and particularly on the transverse processes of the upper cervical vertebræ. The supraorbital region is often involved and the temporal muscle may be the seat of marked thickening. Any portion of the scalp may be thickened or nodular. Chronic glandular enlargement due to irritation produced by proximity to these accumulations is sometimes present and congestive headache may be produced by the circulation being retarded through pressure from these swellings on the veins. This ceases after the dispersement of the enlargements.

DIAGNOSIS

In the diagnosis of a severe paroxysm with agonizing pain in the head and back of the neck, this condition may be mistaken for meningitis, but here fever is present and the indurations and hypersensitiveness absent. In migraine there is frequently a sensitive aura, painful and hypersensitive areas are absent, while nausea and vomiting are present. Hereditary migraine begins in early youth; indurative headache appears in later life, and, finally, migraine is unrelieved by massage. Indurative headache may, of course, develop in persons subject to migrain. Neuralgia and neuritis may result from these thickenings. Cases of hemicrania frequently belong to this group of headaches.

TREATMENT

Under efficient treatment, which will take from one to three months, these enlargements can usually be removed. There are a few cases of induration of such marked cartilaginous consistency and of such long standing that only amelioration can be effected.

Persons in whom there is a tendency to these thickenings should take active exercise in the open air, drink water freely between meals, avoid overfatigue from excessive physical or mental activity and emotional excitement. If a relationship between this condition and uric acid is subsequently proved, the diet should be low in the percentage of purins; it should be nutritious, of easy assimilation and not too abundant; alkaline waters are useful; tea and coffee should be used only sparingly, and alcoholic beverages should be avoided. Internal medication does but little good unless a reconstructive tonic is indicated by general debility. Potassium iodid may give some relief in advanced cases. For a severe acute attack a brisk purgative should be given, a warm bath, hot dry applications to the affected part, and repeated large doses of sodium salicylate, aspirin (acetylsalicylic acid) or salophen (acetylparamidophenol salicylate). The removal of the thickenings will require from one to three months, and the measures given, in the order of their efficiency, are massage, vibration and galvanism. The galvanic current may possibly be made useful, the anode being placed over the affected area and the cathode at an indifferent point. Vibration by means of the electric vibrator helps to remove the thickenings and is conducive to more rapid absorption, but should not be used about the head in cases of arteriosclerosis. The most effectual means of removing these enlargements is without doubt massage. This is at first painful, but after a few treatments the hypersensitiveness disappears. During these treatments a local application of an ointment containing methyl salicylate and capsicum is of some benefit, the former drug acting by absorption and the latter doing good as a counter-irritant. Massage sometimes causes enlargement and tenderness of the lymph glands which receive lymph from the site of the swellings, but this soon subsides.

In all these cases the bowels should be kept active and it is not enough that they be opened daily; they must be emptied; this may be accomplished by the generous use of the laxative mineral waters. The treatment is complete when the swellings have disappeared and tenderness no longer exists.

Few patients are more grateful than those relieved of the sufferings from these agonizing and obstinate headaches.

REPORT OF CASES

I report three cases which, as far as I am able to determine, are the first reported by an American physician. The first history which I give is not from a case of my own, but from a patient whom it was my privilege to examine some weeks ago while he was under treatment.

CASE 1.—*History.*—The patient was a lawyer, aged 40, who had had headaches since boyhood. For the last three years they had become very severe and had occurred as often as once or twice a week with a duration of one or two days. They were frequently brought on by work in the court-room and were so incapacitating that the patient thought at times he should have to relinquish the practice of law. He did not remember that they were affected by damp, wet weather, but recalled that while traveling in England they were very annoying and that he was free from them during a stay in Switzerland. The headaches were largely confined to the two temporal regions.

Examination.—The patient appeared anemic, poorly nourished, and of a color which suggested a sluggish liver. Cartilage-like thickenings were found on the transverse processes of the upper cervical vertebræ, marked enlargements offering considerable resistance at the insertions of the muscles around the base of the skull between the mastoid processes. Swellings were present in the sternomastoid muscles from the level of the thyroid cartilage to their clavicular attachments. Both temporal muscles were involved; they were thickened and very sensitive and it was to these areas that the headaches were largely limited.

Course of Disease.—The patient has now been under treatment every other day for two months, and during this time has only had four attacks, all of which were mild and of short duration. His general appearance has improved; there is an increase in weight and his work is now done without fatigue.

CASE 2.—*History.*—The patient, aged 63, was formerly owner of a business college. He had suffered for years from pain in the back of the head and neck. He said that he had had several attacks of renal colic and frequently noticed a brick-dust sediment in his urine. He was very susceptible to cold and was much affected by damp, chilly weather. He was troubled with stiffness in the back of the neck, where he said he did not have proper use of his muscles. Because of poor health his business was disposed of a few years ago and he then spent some months in California, after which he was somewhat improved.

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Examination.—The patient was rather poorly nourished and anemic. Forcible flexion and twisting of the neck caused intense pain in the back of that region. Areas offering marked resistance and sensitiveness were found over the transverse processes of the upper cervical vertebræ; the occipital portion of the occipitofrontalis muscle was thickened and painful, and there were two enlargements which were very painful on pressure on either side of the spine just below the seventh cervical vertebra.

Case 3.—I have myself suffered from this condition. I was free from headaches until 18 years of age, and since then have really suffered only during the last six years. A dull ache was often present but severe attacks have not occurred oftener than two or three times a month and have been most frequent and painful in the fall. Bad weather has often brought on a headache, as has my being in crowded and poorly ventilated rooms. Occasionally indigestion would be followed by an attack, but there was never any accompanying nausea or vomiting. I have frequently been troubled with a stiff neck and different portions of the trapezius muscle have often been painful on slight movement. My headaches were on the left side and were mainly limited to the temporal and supraorbital regions. For a long time I had noticed that with a headache the left supraorbital region was tender and swollen as well as painful. The left temporal region was always swollen and tender from the zygomatic arch to the temporal ridge. In the course of an attack, this swelling would increase so that the left arm of my glasses would cut a distinct furrow where it crossed this area. Two small patches of exceeding tenderness were present in the auricularis superior muscles just above the ears. During the paroxysms the headaches were constant, severe, and very depressing. They lasted from a few hours to two days and twice I was confined to my bed. Pressure over these areas always gave some relief. Local enlargements were also present causing distinct puffiness of the sternomastoid muscles on a level with the thyroid cartilage. Quick forcible flexion would cause pain at the cephalic insertions of the muscles along the superior curved line of the occipital bone. This line at the base of the scalp was always sensitive to pressure but there was never an ache in this portion of my head. It was not until last summer that I determined the cause of these headaches, since when, through massage and vibration, the swellings and sensitive areas have disappeared, and the pain has ceased.

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CHRONIC "RHEUMATIC" MYOSITIS (MUSKELSCHWIELEN),

WITH CASES SHOWING SOME COMMON ERRORS IN DIAGNOSIS.

BY N. S. YAWGER, M.D.,

ASSISTANT PHYSICIAN IN THE DISPENSARY FOR NERVOUS DISEASES IN THE HOSPITAL OF THE UNIVERSITY OF PENNSYLVANIA.

I firmly believe that the most frequent cause of the so-called "gouty" or "rheumatic" pain is a form of chronic myositis, and surely suffering which is so widespread and which will in most cases yield to appropriate treatment demands our attention. These muscle deposits have been recognised and successfully treated abroad in the Germanic countries for more than 35 years.

At the January meeting of the Philadelphia Neurological Society of this year, in a paper on "Indurative Headache,"[1] I called attention briefly to these general muscular thickenings. While reading the references for that paper I was amazed to learn of the frequency of the development of chronic myositis in muscles other than those of the head and neck, and my attention was also attracted to errors in diagnosis to which this condition has given rise. As early as 1843 Froriep[2] recognised the condition, but the first important contribution to medical literature upon this subject is by Dr. Uno Helleday,[3] a Swede, who, in 1876, wrote extensively about this matter and reported eight cases showing that lesions occur in various parts of the body, all of which he treated successfully. The article finally ends as follows: "The object of these histories and what has gone before is to impress you with the importance of carefully examining the musculature of many cases which present a picture other than that which you usually understand as muscular rheumatism." Gowers,[4] under the heading of "Neuromyositis," writes without being at all definite about the subject, but he evidently recognises that it is important when in closing he says: "The influence of gout, including ancestral gout, is a subject upon which the young practitioner starts with a high degree of scepticism regarding the teaching of his seniors. But year by year his doubts become fewer and fewer, as they are rubbed away, or removed more sharply, by contact with fact." Because of the thoroughness of Gowers's writings, I mention his book to show that this subject has made almost no headway among the English-speaking people since its careful study by the Scandinavians more than 35 years ago.

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[1] Indurative Headache (Schwielenkopfschmerz), with Report of Three Cases, Journal of the American Medical Association, April 24, 1909, vol. lii., p. 1316.

[2] Ein Beitrag zur Pathologie and Therapie des Rheumatismus, Weimar, 1843.

[3] Om Myitis Chronica (Rheumatica), Nordiskt Medicinskt Arkiv, vol. viii., 1876.

[4] Diseases of the Nervous System, P. Blakiston's Son and Co., Philadelphia, 1903, vol. i., p. 134.

Among the causes affecting this condition is heredity, there being a distinct family tendency to these diseased muscles. The effects of climate and weather are most marked. In a case of indurative headache which I have reported the patient found the damp, foggy weather of England very annoying, but he was happily free from suffering while travelling in Switzerland. Chilly and rainy weather often causes the indurations to become painful; it is sufficient to be within the outer limits of storm conditions and not necessarily within the area of rainfall. They may appear at almost any time of life. I have recently observed temporarily infiltrated muscles in the neck of a child of two years and they are common in old age. While probably no voluntary muscle enjoys an immunity there are sites of predilection, the most common of which is in the muscles of the head and neck. This, I believe, is due to the exposure through lack of covering, to which this part of the body is necessarily subjected. The gluteal, lumbar and deltoid regions, and calf muscles are often involved. The muscles may be invaded at their points of insertion, as in those attached to the superior curved line of the occipital bone and those inserting into the processes of the upper cervical vertebræ, or the infiltrations may appear within the bodies of the muscles as is frequently seen in the abdominal walls, and in the sterno-mastoid muscles near the level of the thyroid cartilage. At times the infiltration extends to the periosteum to which the muscles are attached and also to the fascia.

The pathology of these diseased muscles is largely hypothetical for the reason that people do not die from this condition. By some it has been thought to be of nervous origin, but Vogel, quoted by Bum,[5] in sections from rheumatic muscle found thickening and adhesion of the neurolemma to the nerve branches supplying the muscles, a finding which does not strengthen the neurogenous hypothesis of this condition. Oppolzer,[6] from his studies, believes the lesions are not inflammatory in character, a view which does not coincide with that of Helleday. The muscle infiltration may be due to the deposition of an excess of the salts of uric acid or an allied substance, with the later formation of connective tissue. I have known vigorous massage treatment of a patient with extensive indurations to be followed by an attack of acute muscular rheumatism. It seems probable that this formation of indurations is a manifestation of a metabolic disturbance autotoxic in character. At all events it appears that as soon as good circulation is established relief is at once experienced. Whatever this muscle poison is, many of its subjects have had bilious attacks in childhood, and in advanced life they often suffer from arteriosclerosis.

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[5] Handbuch der Massage und Heilgymnastik, Urban und Schwarzenberg, Berlin, 1902, p. 293.

These infiltrations or myisitides (the Germans prefer myitides) may "make up" over night; they are frequently absorbed but tend to recur. After appearing again and again at the same point they finally become chronic and then vigorous treatment is necessary for their removal. The enlargements present a different feeling upon palpation, depending largely upon their duration; the longer they exist the firmer they become and the more resistant they are to treatment. Those of recent development are known as *swellings* and feel doughy to the touch; others because of their firmer consistency are spoken of as offering resistance; and finally those of long standing show cartilage-like thickenings and are known as *indurations*. They also vary in size and in the intensity of the pain which they occasion. There may be a diffuse and extensive infiltration which causes but little inconvenience and which may not manifest itself except during action of the involved muscle, as is seen in the milder cases of lumbago. Again, small patches may give rise to frequent and excruciating pain and this is not unusual in the abdominal muscles, where shot-like enlargements are often found.

It has seemed in what I can gather from the literature and by my own personal experience that these deposits may develop under the following conditions: in muscles that are overworked, as in some cases of writer's cramp[7] and other occupation neuroses; in those that are not sufficiently exercised; in muscles that are much exposed, as in those of the head and neck; and, finally, where an injury has been sustained, which is frequently the case in the back. The symptoms are those of pain which may be sharp and cramp-like, or there may be an aching sensation upon use of the diseased tissue. Instead of pain there is at

[6] Allgemeine Wiener Medicinische Zeitung, v. 27, 1867.

[7] Wide: Handbook of Medical and Orthopædic Gymnastics, Funk and Wagnalls Company, New York, 1903, p. 277.

times paræsthesia at some distance from the deposit, as in a patient seen by Norström[8] in Paris, who had for years complained of the sensations of cold, numbness, formication, &c., in the thumb and two adjoining fingers of the right hand, and which upon examination presented a big lump in the deltoid muscle which pressed upon the radial nerve without giving any special abnormal manifestation in the muscle itself. There is often muscle insufficiency. Upon pressure made with a friction movement there is tenderness, but firm and steady pressure often affords relief. If the swollen muscle impinges upon a nerve trunk or nerve fibre a true neuralgic pain will result. There may be diminished or absent elasticity; finally, the presence of a palpable thickening in the muscle; fever and redness are absent. There are often associated symptoms in various parts of the body, some of which are depression of spirits, mental torpor, unnatural drowsiness, marked susceptibility to cold, torpidity of the liver, indigestion, painful spasms of the calf muscles, paræsthesia, general myasthenia, pyorrhœa alveolaris, and hypersensitiveness of the teeth.

To those familiar with muscle conditions I know of but one lesion that is likely to be mistaken for an induration, and that is a gumma. Indurations are exceedingly common; there is a history of chronic rheumatic pains; as a rule they are less sharply defined; there is pain upon pressure with friction, and they yield to massage treatment. Gummata in muscles are relatively rare, are not usually painful, are attended by other evidence of syphilis, and disappear under antispecific medication.

The mistakes in diagnosis to which these rheumatic deposits give rise are very numerous. Many cases of cephalalgia are of this variety;[9] involvement of the intercostal muscles has been taken for intercostal neuralgia and pleurisy; in the abdominal wall for acute and chronic appendicitis, gastric ulcer, abdominal and pelvic adhesions, calculus of the kidney, and floating kidney; involvement of the gluteal region for sciatica and hip-joint disease; in the neck they have been mistaken for swollen glands; the frequently complained of growing pains in children I believe are due to this condition, since the healthy growth of a child should not be painful.

I shall allude briefly to a case reported by Kleen[10] which was at first thought to be one of gastric ulcer. The patient was a female, aged 23 years, single, who for more than a year had suffered from pain circumscribed at a point over the gastric region a little to the left of the median line. The pain was constant but worse after meals. There were other slight symptoms of indigestion. Upon palpation a distinct difference in the consistency of the upper portions of the recti abdominales muscles was made out, the left being the harder and the more extensively infiltrated. At this area careful lateral pressure caused sharp pain which evidently had its origin in the muscle. When she endeavoured to rise without the assistance of her hands the pain was intensified at this point. The patient recovered under less than a month's massage treatment.

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[8] Chronic Headache and its Treatment by Massage, New York Medical Journal, 1905, vol. lxxxii., p. 956.

[9] Edinger: Diseases of the Nervous System, Appleton and Co., New York, 1908, p. 863.

[10] Handbook of Massage, P. Blakiston's Son and Co., Philadelphia, 1892, p. 95.

I have myself encountered indurations in the abdominal muscles and know of a Philadelphia woman who for a long time had suffered from sharp attacks of pain in the abdominal region. During a trip abroad she had an attack of unusual severity. A physician was summoned, a diagnosis of appendicitis was made, and immediate operation was advised. Having recovered from a number of milder attacks the patient was not sufficiently alarmed to submit to the suggested surgical interference, and in a day or so the pain subsided. Upon her return to Philadelphia her condition was recognised as being due to deposits in the abdominal muscles. Through a course of massage the diseased muscles were restored to normal, and while she still has her appendix she no longer has the abdominal pains.

Moderate distension of the abdomen with gas is very common, and with the abdominal muscles in this indurated condition it takes but slight distension to cause stretching of the diseased muscle fibres, thereby causing pain. In some of the cases where normal appendices are found at operation it is the abdominal muscles that are at fault. I have recently examined the abdomen of a physician while he was having an attack of colicky pain. He was subject to mild attacks of this kind and stated that some time before he believed that he had an attack of appendicitis, but that an examining surgeon advised against operation. I found his abdomen slightly tympanitic and the abdominal muscles infiltrated and very tender. The pain was relieved by firm, steady pressure. Some of the muscles of the head and neck were hypersensitive and he often suffered from headache. Within a few hours the distension subsided and the pain ceased. I think it very unlikely that his appendix had at any time caused the abdominal pain.

I am aware of the incredulous attitude of orthopædic surgeons towards a rheumatic condition about the hip-joint of a child, but the condition surely exists at times, and there are a number of cases in literature where rheumatism has been mistaken for incipient tuberculosis of this part and the child has been placed upon extension, while a later diagnosis was followed by proper treatment and prompt recovery. This may account for cases of hip-joint disease reported as cured without shortening and with perfect motion. Many cases of early hip-joint disease are diagnosed as something else, and the fact remains that a few cases of lameness with rigidity of the muscles about the hip and pain around the hip and knee are rheumatic and should be recognized and treated as such.

The following case was referred to me by Dr. Ralph Butler in April of this year. The patient was a dressmaker, aged 38 years, in whom the present trouble began 14 years ago with an intermittent grumbling pain in the right lower jaw. Six weeks later she had a severe attack with pain extending into the right ear and back part of the right side of the tongue. The year following the attacks became frequent and quite severe. At times several months would pass without paroxysms; again they might occur every five or ten days and persist for days or even weeks. The neuralgia would often begin with frequent and mild attacks, then recur more often and be of greater intensity. At times when asleep she would be awakened by such an agonising attack that she would cry out with pain. She has walked the floor for hours with her hand pressed as forcibly as possible against her face. For a week at a time her face has been so painful and stiff that she had to limit herself to a liquid diet. There were occasions when it was almost impossible to avoid provoking attacks, they being caused by passing a comb ever so gently through the hair, the slightest breath of cold air, brushing the teeth, putting out the tongue, or even attempting to speak. The paroxysms would start with just a few twinges of pain; these would increase, and then the most agonising suffering would ensue. Attacks would last for almost a minute, recur again and again, and finally cease, leaving the patient almost exhausted. During the most severe attacks the affected side of the face would suddenly become flushed and the eyeball injected; this would pass away with the paroxysm. During the most severe attacks the pain would extend into the orbital cavity. Her oculist was consulted yearly in the hope of obtaining relief, six teeth were extracted, but all without improvement. Three years after the beginning of the attacks the facial muscles began to twitch; this was relieved by an injection of alcohol. A year ago a neurologist was consulted, who said that the Gasserian ganglion should be removed at once. In addition to the neuralgia there had been headaches, which were very severe and of late had become almost constant. There was much pain in the back of the neck and at times the scalp was very sensitive. She had frequently suffered from rheumatic pains in different parts of the body, especially in the right arm and about the right hip, where infiltrated areas were present. The neuralgia, headaches, and bodily pains were all worse in damp and stormy weather. A year ago she was obliged to give up her work for six months, and this spring her suffering was just as great.

On examination the patient was anæmic, with an anxious expression, and presented the appearance of one who had undergone great suffering. The scalp was thickened and hypersensitive, with especially painful areas over the trapezius muscle and in the supraorbital region on the right side; these areas were indurated, as were most of the trapezius and other muscles attached to the occipital bone. There was also involvement of the muscles on either side of the neck, especially at their attachments to the transverse processes of the upper cervical vertebræ. The most sensitive part of the face was along the upper lip just below the nose. It would take but the slightest touch at this point to provoke a paroxysm. The small facial muscles were thickened and painful upon the affected side.

I made a diagnosis of trifacial neuralgia (tic douloureux) of peripheral origin, involving the three divisions of the nerve, and indurative headache. Local treatment in the form of manual massage and rapid vibration delivered by an electric motor was advised. After a few treatments the spontaneous attacks of neuralgia ceased, though they could be provoked by pressure over the hypersensitive area of the face. Three treatments per week for two months were followed by cessation of both headache and neuralgia. The treatment was continued for a month longer since some of the involved muscles were still quite sensitive, and it was evident that absorption had not been complete. There was no internal medication except a gastric tonic for two weeks and the free use of an alkaline laxative water during the first month. I do not yet report this case as one permanently cured, but the disease is certainly arrested, and I hope the good hygienic life under which the patient now lives may prevent the further formation of indurations.

A STUDY OF THE AXIS-CYLINDERS IN FIVE CASES OF GLIOMA CEREBRI

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It is not infrequently observed in a variety of neuropathological conditions, such as multiple sclerosis, tuberculoma, areas of compression and gliomata, that the necropsy reveals a much more extensive process than could have been even suspected from a most thorough clinical examination. This disproportion between the diseased area and the symptoms induced is, perhaps, more pronounced in gliomata than in any other condition, and it is to this pathological state that my attention has been directed.

It is well known that gliomata may exist for months or even years without causing either general or localizing symptoms of brain tumor. This might be conceivable where the tumor is situated in a silent area. According to Brush (1), however, such may be the case even though a localizing center be involved, and several cases have been reported in which no abnormal condition was manifested during life. On the other hand, those cases which do make themselves obvious to the clinician, occasionally show so few symptoms or else they have existed for so short a time that one is quite surprised when the pathologist presents him with a specimen in which one or more lobes, or a greater part of the brain stem is markedly involved; while a still more interesting feature is the occasional absence of degenerative changes above or below the lesion. Considering the ordinarily slow growth of such tumors, the questions naturally arise: How are we to explain this persistence of function and absence of degenerative changes where the cerebral tissue is extensively involved; and how are we to account for the apparently rapid cases which show a pathological state similar to those existing for months or even years?

The answer to the first question will naturally include the second, for if we can explain the persistence of function in simple uncomplicated gliomata, and if we admit that they can exist for years without producing symptoms; then the so-called rapid cases might easily be due to some intercurrent condition, such as hemorrhage, areas of softening or myxomatous changes in a preexisting gliomatous mass which had hitherto been quiescent. However this may be, it shall be my effort to demonstrate that in simple cerebral gliomata persistence of function and absence of degenerative changes in the spinal cord may be explained upon a definite histological basis.

Let us review first the theories which have been offered in explanation of these facts.

From a study of the few cases which have been examined with this object in view, two rather theoretical explanations may be formulated. The first might be called the theory of displacement and is well illustrated in the case reported by Schorstein and Walton (2), who studied a glioma of the pons in a male child four years of age. Examination revealed slight spasticity of the lower extremities, more marked in the right side, and some weakness in the right arm. The left arm was practically normal. Increased knee jerks, no ankle clonus, and Babinski was negative. Duration—two months. There was found at autopsy a tumor measuring 2 x 1½ inches, situated in the left half of the pons and medulla, with slight invasion of the right half. The new growth passed gradually into the surrounding normal tissue, and showed in its center an area of softening. Microscopic sections were made from each half of the pons and from six different regions of the spinal cord. Those from the pons were stained with polychrome methylene blue. The right half showed some normal pontal tissue with many disarranged pyramidal fibers in the midst of a cellular tumor. The left half showed no normal pontal tissue, and an apparent absence of the transverse pontal and pyramidal fibers. Sections from the cord were stained with Busch's fluid, and hemalum and Van Gieson. Those stained by the former method showed no degenerative changes in any part, while the Van Gieson stain revealed the usual number of axis-cylinders.

Although the clinical findings and pathological condition at the site of the lesion were somewhat at variance, it is the absence of degenerative changes in the cord which constitutes the striking feature in this case. How could there be complete absence of pyramidal fibers in the left pontal region, with decided evidence of functional disturbance, and yet no sign of degeneration in the spinal cord? The author's explanation was that the left pyramidal fibers had been "pushed aside" without an interruption in continuity, although functionally inadequate. This seems to be a purely theoretical assumption, for it appears likely that had the fibers of the left side been displaced into the right pontal area, then this right half should have shown a definite increase in the number of pyramidal fasciculi, for it would have contained not only the fibers normally present, but those contributed from the left half. This should have been apparent to the naked eye on cut surface, yet no mention was made of such an observation. On the other hand, what has become of the transverse pontal fibers of the left half, which, it will be remembered, were entirely absent; and why were there no cerebellar symptoms pointing to their apparent destruction? Unfortunately no sections were made from the middle cerebellar peduncles and no evidence was adduced to account for the absence of the left pontal fibers. Furthermore, if this idea of displacement without anatomical interruption or other evidence of histological changes in the fibers be correct, why were the symptoms confined to the pyramidal system; none from the pontal fibers in the very heart of the tumor; and the whole symptom complex of only two months' duration? All of this may be due to pressure changes; yet why were there no pressure symptoms during the process of displacement, for surely the tumor did not attain such dimensions, push all the fasciculi to the right side and destroy the left pontal fibers in only two months.

In Davis' (3) case, for instance, there was found a glioma of the left frontal region, measuring 11 x 9 x 6 cm., which had pushed the thalamus, caudate nucleus and internal capsule to the right and posteriorly. The symptoms, which were "very slight," were of only two months' duration. Of course, this may be explained in two ways—one may say it was a very rapidly growing tumor or else it was a more gradual process which, after reaching certain dimensions, began to exert pressure changes, and that the first symptoms were evidences of this beginning pressure, which in two months attained such proportions as were found post-mortem. But evidently a tumor of the frontal region would have to attain considerable size before producing the displacement found in this case, and if size be any indication of chronicity, must have existed for some time. What, then, was the condition of affairs during the incipient stage? Surely there must have been some pressure exerted, and Marinesco and Minea (4) have shown that even very slight pressure of the shortest duration experimentally produced is sufficient to cause definite histological changes in the nervous system. Why, then, were the symptoms delayed until such marked displacement occurred? No report was made upon the spinal cord or the internal capsule.

Other cases might be reviewed in which there have been marked evidences of pressure changes, but they have been so incompletely studied from a pathological point of view that an abstract of them is scarcely worth while. In all the usual occurrence of a large tumor and relatively few symptoms were observed, but no report was made upon the condition of the nerve fibers involved by the new growth.

While it is not my intention to deny the occurrence of decided pressure symptoms and evidence of displacement in gliomata, it does not seem that such facts satisfactorily account for all the conditions observed. The objections, then, to such an explanation are: First, the failure to demonstrate that all the fasciculi are "pushed aside"; second, that it does not account for the absence of symptoms during the earlier stages when the infiltrating cells are in intimate contact with and no doubt exerting some pressure upon individual fibers; and lastly, it is thoroughly inadequate, in those cases where the entire pons or internal capsule shows no normal fasciculi upon macroscopic examination, nor any evidence of displacement.

The second and more widely accepted view rests upon the infiltrating nature of gliomata and the presence of medullated fibers in the tumor substance. Such observations led to the conclusion that the persistence of function and absence of degenerative changes below the lesion were due to these retained nerve fibers which passed through the tumor without interruption.

Sir William Gowers (5), in writing of gliomata, says: "Some infiltrate without displacing the cerebral substance, others displace rather than invade. From their infiltrating character they usually cause simple enlargement without displacement or compression of adjacent parts," and "sometimes nerve fibers persist and retain their conducting power in a part which is almost entirely infiltrated with the new growth." This seems to be the position taken by the majority of investigators who have demonstrated the presence of nerve fibers in gliomatous tissue. However, the conclusion that the fibers pass through without interruption does not appear to have been definitely proven, since most of the observations were made from small fragments of the tumor without any report upon the region above or below the new growth. Although one might find a few scattered fibers in a single portion of a tumor this is not sufficient to prove that they pass through as continuous neurones, for sections made from a greater depth in the same area or from the central portion of the tumor might show an entire absence of fibers, and no case was reported in which the entire neoplasm had been sectioned. Furthermore, it is a common observation that the usual medullary stains reveal a decreasing number of fibers from the periphery toward the center, while certain gliomatous areas show an entire absence of medullated fibers.

Bassoe's (6) monograph is interesting in this connection. In one of his three cases there was a large glioma of the left temporal and occipital lobes. The tumor was quite vascular and contained hemorrhagic areas with softening and necrosis. There was no mention of displacement of surrounding parts. The Walters-Kaes myelin stain showed numerous medullated fibers "passing through" the tumor. No degeneration was found in the spinal cord. Stain not mentioned; presumably the Walters-Kaes method was used. It is not quite clear as to what the author means, when he says numerous fibers were "passing through" the tumor. If he intends to convey the idea that they were passing through that portion of the tumor he examined, the point is quite admissible. On the other hand, if he takes the position that, since fibers were found in the tumor and no degeneration was observed in the spinal cord, because of this fact, then it seems that the deduction is insufficiently proven, for one would not expect to find secondary spinal degeneration from a lesion confined to the temporal and occipital lobes.

Although these observations are extremely suggestive, the number of medullated fibers, which can be demonstrated by the usual myeline stains, are so few that provided they did retain their continuity, their number is not sufficient to account for the functional persistence and absence of degenerative changes in associated areas.

Attention was next directed to the axis-cylinders. A great deal of work has been done upon the intra-cellular fibrils in a variety of normal and pathological conditions, while surprisingly little attention has been paid to the extra-cellular fibrils of the axis-cylinders. The first contributions appeared simultaneously by Bartels (7) and Bielschowsky (8), both of whom studied the condition of the axis-cylinders in multiple sclerosis, using the Cajal and Bielschowsky methods. They found everywhere in the sclerotic areas a surprising number of axis-cylinders of normal direction and appearance. Later, Raubetschek (12) in studying a tuberculoma of the spinal cord by Bielschowsky's method was able to demonstrate the presence of axis-cylinders in the diseased area, but made no report upon the conditions above or below the lesion, nor did he use the Weigert method. All this, naturally, led to some speculation concerning gliomata. Strobe (13) had already shown the presence of medullated fibers in gliomata to be a fairly constant feature; that they increased in number from the center towards the periphery and frequently showed marked degenerative changes. Their number, however, was entirely too small to explain the marked functional persistence, and in order to be consistent, it was commonly held that a great many more fibers could be demonstrated with a reliable axis-cylinder stain.

Bielschowsky (9) had already produced the required stain in 1902, and to him also belongs the credit of having first applied it to the study of gliomata, for in 1904 (10) appeared his first article upon the axis-cylinders in a glioma of the pons. The Weigert preparation showed a number of medullated fibers at the periphery of the tumor, while there were only a few fragments in the center. The author's silver impregnation, however, revealed a great many axis-cylinders in all parts of the tumor. These were considered as persisting fibers and resembled very closely those found in multiple sclerosis.

The following year or so was marked by a lively interest in the extra-cellular fibrils and they were studied in a variety of conditions, such as carcinoma, sarcoma, areas of compression and gliomata; and were found to be present in each case in variable numbers, and in all stages of histologic preservation.

In 1906 appeared another communication by Bielschowsky (11) in which he studied two more gliomata. The first tumor was situated in the left frontal lobe, had advanced upon the corpus striatum, and contained in its posterior portion a few small hemorrhages. Duration—two years. Sections were cut so as to include both center and periphery of the tumor, that is, radially to its center. The technical methods used were hemotoxylin and eosin, Van Gieson, Wiegert, the author's silver impregnation and Benda's glia stain. To the unaided eye the Weigert preparation showed a marked difference between the center and the periphery; the center was golden yellow, while the periphery was dark blue from the presence of numerous medullated fibers. The contrast, however, was not so striking when examined microscopically, and the central portion showed several pale, swollen medullated fibers of irregular contour and many myelin droplets. In the silver impregnation the whole section stained more uniformly and the transition from center to periphery was quite gradual. Microscopically, there were decided histologic changes in the axis-cylinders, considered by the author to be regenerative in character.

In the second case the tumor was situated in the left temporal lobe and extended into the inferior horn of the lateral ventricle. The new growth was quite vascular, contained a small hemorrhage in the center, and numerous fibers from the surrounding brain tissue could be seen radiating towards its center. The microscopic findings were practically the same as in the above case except for the intimate relation of the new fibers to the blood vessels.

In neither case was any report made upon the spinal cord, and the author's attention was more fixed upon the regenerative processes than upon the possibility of anatomical continuity.

Since these studies were published the interesting work by

Herxheimer and Gierlich (14) on neuro-fibrils has made its appearance. The authors have employed Bielschowsky's silver method in normal and pathological conditions with most excellent results. Two gliomata were examined by them, but in both cases the tumor alone was studied. Sections were made from the center and the periphery. The results were practically the same as were found in Bielschowsky's studies except that the axis-cylinders were more nearly normal. It was also observed that in the hemorrhagic and necrotic areas the fibrils were greatly diminished or entirely absent.

While such studies leave no doubt that in these conditions nerve fibers may be found in far greater numbers by a satisfactory axis-cylinder impregnation than by any of the myelin stains, or the usual axis-cylinder stains, it does not appear to be demonstrated that these fibers pass through the new growth without interruption.

Thus it was that my studies were undertaken in order to confirm the result already obtained and to demonstrate, if possible, the continuous course of the persisting fibers.

In all, five gliomata were studied. Three are situated in the left cerebral hemisphere, the fourth in the right hemisphere, and the fifth in the pons and medulla. In each case a small piece of tissue was taken from the center of the tumor, and cut by the freezing method into sections varying from ten to fifteen microns each. All sections from each tumor were cut from the same block of tissue. The staining methods used were: hemalum-acid fuchsin, Weigert's and Bielschowsky's silver impregnation for neuro-fibrils.

In the first case the tumor is located in the left occipital and temporal lobes, and measures eight centimeters in its antero-posterior diameter. There are several small hemorrhages scattered throughout and the blood vessels are numerous. The internal capsule is apparently normal. A small piece of tissue was taken from the center of the tumor and from the entire posterior limb of the internal capsule which was divided into anterior, middle and posterior thirds. Sections from each specimen were stained by the three methods employed.

The Tumor.—Hemalum-acid fuchsin showed an extremely vascular tissue made up of cells with round and oval nuclei, and many neuroglia fibers. No nerve fibers are observed in any part of the section.

Weigert Section.—The cellular and vascular characters can still be recognized, and a few swollen, beaded, medullary fragments can be seen (Fig. 1). In the center of the section, where the nerve fibers seem to be least numerous, twenty-seven fragments were counted. Oc. 1. Obj. ⅔. Whereas, if the ⅙ obj. were focused on the same area no medullary fragment could be seen. At the periphery of the section numerous medullary fibers are observed with each magnification.

Bielschowsky Section.—The character of the tissue can still be discerned and many axis-cylinders are seen in all parts of the section (Fig. 2). In that portion of the section which showed the least number of fibers, sixty-eight were counted with the ⅔ obj., and in the same field twenty-four were counted with the ⅙ obj. The axis-cylinders had no definite direction, varied considerably in size, and showed decided evidences of degeneration in certain areas.

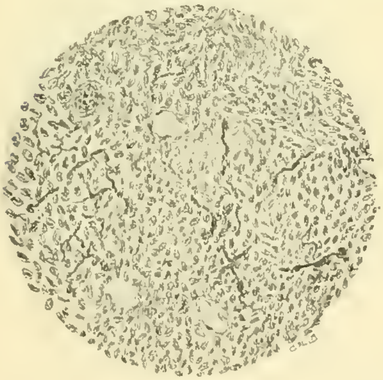


FIG. 1. Weigert preparation from substance of tumor, Case I, showing a few medullated fragments. Oc. 1, Ob. ⅔.

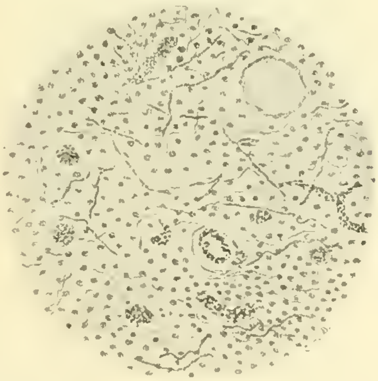


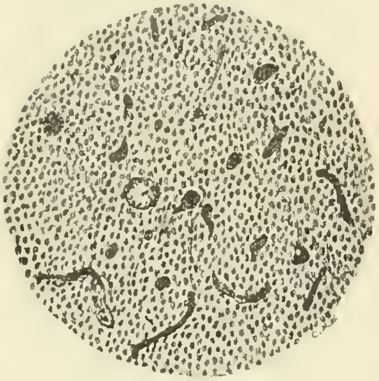
FIG. 2. Bielschowsky preparation from same block of tissue as Fig. 1, showing many axis-cylinders. Oc. 1, Ob. ⅔.

The Internal Capsule.—The anterior third appears to be normal by each of the three stains. The middle and posterior thirds show degenerative changes by all three methods, and a small capillary hemorrhage is observed in the posterior third, and some proliferation of the neuroglia cells. In the degenerated areas a great many more axis-cylinders are observed in the silver preparation than in either of the other two stains. The hemorrhagic area shows a complete absence of medullated fibers in the

Weigert preparation, while the silver preparation shows many fibers in this area.

Although the number of persisting fibers is comparatively small in this case, such has been the usual observation in hemorrhagic gliomata and in areas of secondary softening (14).

In the second case there is a large glioma situated in the left occipital, parietal and temporal lobes, with extension into the left cerebral crus. In its center there is an area of softening. The specimen of tissue taken from the center of the tumor was treated in the manner described and shows in each case such a marked resemblance to the one just reported that one description will serve for both. The second tumor, however, contains a great many more axis-cylinders in the silver preparation than are found in the first case.



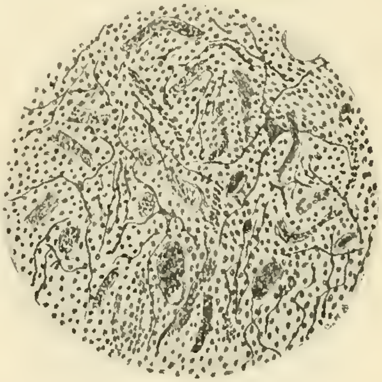


FIG. 3. Weigert preparation from substance of tumor, Case 3, showing an entire absence of fibers. Oc. 1, Ob. $\frac{2}{3}$.

Fig. 4. Bielschowsky preparation from same block of tissue as Fig. 3, showing numerous axis-cylinders and two ganglion cells. Oc. 1, Ob. $\frac{2}{3}$. No part of this section was free from fibers.

The third tumor involved the right occipital and temporal lobes and is quite hemorrhagic. The internal capsule and thalamus are greatly displaced to the left. The hemalum-acid fuchsin stain is similar to the above two cases. The Weigert preparation shows a complete absence of medullated fibers, while the silver method reveals a great many axis-cylinders (Figs. 3 and 4).

The fourth tumor is extremely large, is gelatinous in places, and is situated in the left cerebral hemisphere. Portions of the occipital and temporal lobes, the internal capsule, thalamus and lenticular nucleus are involved; and there is decided displacement of the surrounding tissue. As in the third tumor there is complete absence of fibers in the Weigert preparation. The silver impregnation shows a number of axis-cylinders, most of which appear quite normal. (This case has been reported by Dr. Charles K. Mills (15), who studied it from the physiological and pathological aspects. His article also includes a gross pathological description of the tumor by Dr. William G. Spiller.)

The fifth case is a tumor of the pons. The basilar portion of the pons is considerably enlarged and presents a lobulated appearance due to the presence of a soft gelatinous growth. The tumor is more pronounced on the left side, where it measures 4.5 c.m. antero-posteriorly, while on the right side it measures only 2.5 c.m. The left middle cerebellar peduncle is involved, and anteriorly the new growth extends into the interpeduncular fossa, where it encroaches upon the left cerebral crus. The new growth in each half of the pons becomes continuous superior to the basilar artery and at the upper border of the pons completely surrounds the artery. On the left side no transverse pontal fibers are observed, while on the right side a few of these fibers may be seen where the middle peduncle enters the cerebellum. Transverse sections through the cerebral hemispheres show decided involvement of the left internal capsule and cerebral crus.

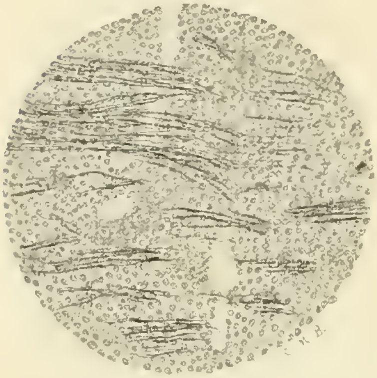
A transverse section through the middle of the pons shows no normal pontal tissue in either half, and the basal and tegmental areas are equally involved. The pyramidal fibers are apparently absent in both sides, having been replaced by extremely gelatinous and translucent tissue. The superficial transverse pontal fibers are entirely absent, while the deep fibers are considerably swollen. The left half of the pons is somewhat larger than the right, and has deflected the aqueduct of Sylvius to the right. There is no evidence of displacement in the basilar portion; but rather a condition of general enlargement. The anterior pyramids appear normal in transverse sections.

The patient was a female and had had symptoms for about one year. The optic discs were greatly swollen; there was slight ataxia; exaggerated reflexes in right arm and leg; no ankle clonus; and Babinski was positive.

A small piece of tissue was taken from the left half of the pons in an area which would normally contain many pyramidal fasciculi. This together with a portion of the two anterior pyamids was sectioned longitudinally and stained as in the preceding cases.

The Tumor.—The hemalum fuchsin stain reveals the gliomatous character. The tissue is not very vascular and there is no positive evidence of nerve fibers.

The Weigert preparation shows a surprising number of medullated fibers normally directed and fairly well preserved (Fig. 5). They do not, however, extend entirely across the section and some of them are swollen and beaded. Areas are also seen in which no medullary substance is present.



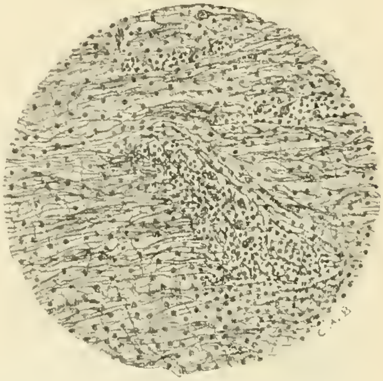
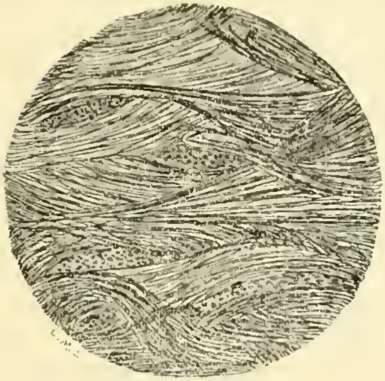


Fig. 5. Weigert stain. Longitudinal section from midst of pontal tumor, showing medullated fibers and areas entirely devoid of fibers. Oc. 1, Ob. $\frac{2}{3}$. Case 5.

Fig. 6. Bielschowsky preparation, from same block of tissue as Fig. 5, showing countless normal axis-cylinders and a few ganglion cells. This is a fair representation of the entire section. Oc. 1, Ob. $\frac{2}{3}$.

The Bielschowsky section shows innumerable axis-cylinders normally directed and surrounded by the infiltrating cells. What astonishes one most is their almost perfect state of preservation (Fig. 6). The fibers are arranged in definite bundles, and numbers of them are seen extending entirely across the microscopic field. There is no evidence of fragmentation, and only occasionally do they show swelling or disintegrative changes. *No part* of the section is free from fibers. Their normal appearance is most interesting since the cases hitherto reported have all shown decided structural changes.

The Pyramids.—The two pyramids are so similar that one description will serve for both. Figs. 7 and 8 represent the left anterior pyramid stained by the Weigert and Bielschowsky methods respectively. In neither case are there definite degenerative changes. The Weigert preparation, in particular, is quite normal looking, the fibers show no swelling or beading, and are present in sufficient numbers. The silver preparation, however, shows some swelling of the axis-cylinders in places. There is no fragmentation and no part of the section is devoid of fibers. A transverse section of the anterior pyramids stained by the Weigert method was studied from a preparation belonging to Dr. Spiller, which he kindly let me use. There was no degeneration in either pyramid.



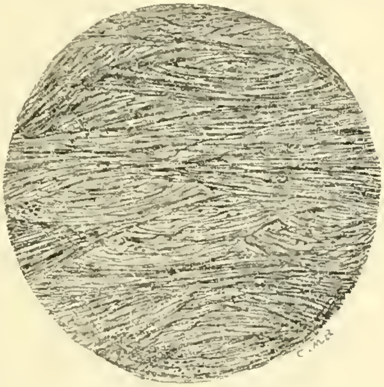


FIG. 7. Weigert preparation from left anterior pyramid cut longitudinally. Case 5. Oc. 1, Ob. $\frac{2}{3}$.

FIG. 8. Bielschowsky preparation from same block of tissue as Fig. 7. Oc. 1, Ob. $\frac{2}{3}$.

For the sake of completeness and to determine whether there might be some "reaction à distance," sections were made from the right and left paracentral lobules in order to study the Betz cells. They were stained by the Bielschowsky and thionin methods. There was some central chromotolysis in the thionin preparation, though not to a marked degree, and the intracellular fibrils more disintegrating in a few cells. Considering the age of the patient and the increased cerebral pressure with probable circulatory disturbances, such observations are of little value. At any rate, the majority of the cells were perfectly normal.

The first criticism from inquiring minds will naturally be concerning the silver preparations. Are all the fibers found in this case, in particular, really axis-cylinders, or may not some of them be neuroglia fibers, connective tissue, or even artefacts? Bielschowsky does not claim to have furnished an exclusive axis-cylinder stain, and states that in his own hands his method does impregnate neuroglia and connective tissue. The distinction, however, between these different elements is quite easily made, and it is often possible to obtain specimens in which the neuroglia fibers are not at all impregnated, a condition which was obtained in a number of my preparations. In case the neuroglia fibers are apparent, they never stain so intensely as the axis cylinders; are as a rule much smaller; have a more wavy contour; do not arrange themselves in bundles; and are not found running uninterruptedly through the entire section.

SUMMARY AND CONCLUSIONS

1. In three of my five cases nerve fibers were found in the substance of gliomatous tissue by both Weigert and Bielschowsky methods.

2. Two of the cases showed an entire absence of fibers, or even myelin droplets by the Weigert method, while unmistakable axis-cylinders could be seen in the silver preparation made from the same blocks of tissue. This is due, no doubt, to the greater vulnerability of the myelin sheath and its early disappearance. Thus there is certainly not a very intimate relationship between this sheath and the axis-cylinder, and Brodmann (16) maintains that there is no relation between myelinization and the formation of fibrils. Consequently, in such cases studied by the Weigert method alone, the absence of fibers in the tumor, and the failure to find degenerations in the spinal cord led to the most obvious conclusion that the fibers had been pushed aside.

3. In all five cases a great many more nerve fibers (axis-cylinders) were found by the silver method than in the Weigert preparation.

4. In the fifth case the entire pons was involved, and there was no evidence of the pyramidal fasciculi upon gross examination, yet a section from the tumor substance treated by the Bielschowsky method showed an enormous number of normal axis-cylinders; the anterior pyramids showed no degeneration in either longitudinal or transverse sections; and the Betz cells in the greater part were quite normal. Considering, then, the loca-

tion of this tumor, its long duration, the absence of marked symptoms pointing to upper segment involvement, and the microscopic findings, it seems reasonable to conclude that not only are nerve fibers retained in gliomatous tissue but that a large number persist as uninterrupted axones, passing through the tumor substance.

In conclusion, I wish to express my gratefulness to Dr. William G. Spiller, of the University of Pennsylvania, for placing the material at my disposal and for his many helpful suggestions.

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THE RELATION BETWEEN ANEURYSM OF THE AORTA AND TABES DORSALIS; WITH THE REPORT OF A CASE.*

By **PAUL H. BIKLÉ, M.D.,**

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LESSER[1] has recently called attention to the fact that in approximately twenty per cent. of the cases of tabes dorsalis which he has observed, he has found aneurysm of the aorta. This not infrequent occurrence of the two conditions has suggested the question whether, in lieu of the unquestionably frequent dependence of each on syphilis as a causative agent, there may not be in tabes itself a peculiar toxine or condition which exerts an influence on the arterial walls, affecting them in some way which renders them less able than normally to withstand intraarterial pressure; or whether the intraarterial pressure which, according to Heitz and Norero[2], is determined by gastric crises, and particularly by the lightning pains, may be a factor in the production of aneurysms. In other words, is the occurrence of the two affections in the same patient merely a coincidence, or shall we find in such cases tabes itself, or a toxin peculiar to the two conditions to be the underlying cause of the aneurysm? Lesser suggests the possibility of such a dependence.

*From the Laboratory of Neuropathology, University of Pennsylvania. Read before the Philadelphia Neurological Society, February 27, 1909

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Arterial disease is manifested chiefly either in a generalized fibrosis of the vessel walls or in a localized circumscribed nodular form, characterized by the development of scattered patches of atheroma and calcification of the arterial coats. In both instances the elasticity of the vessel wall suffers, but in the nodular form there is less expansibility than in the other, where there is simply a fibrosis. It naturally follows that the less expansile the vessel, the more prone it is to rupture when intraarterial pressure is increased, while under the same conditions, if the pressure is great enough to cause any damage, the more expansile form will more probably permanently dilate than rupture. This fibrous form is, according to Flatau, Jacobsohn, Minor,[3] and others, the variety of arteriosclerosis caused by syphilis, while the nodular sort, associated with calcareous deposit, is due to old age, chronic alcoholism, and plumbism, prolonged physical exertion, exposure, and other similar conditions.

There can be no doubt that syphilis is one of the most frequent causes of aneurysms of the aorta, but wide differences exist in the opinions of writers concerning the degree of the frequency of its relation to this form of vascular disease. The extremes are represented on the one hand, by M. Schmidt, who finds syphilis present in 29 per cent. of cases, and on the other, by Drummond, who believes that lues is responsible for aortic aneurysms in every instance. Debove[4] also shares the latter view, while Gerhardt, more conservatively, regards 53 per cent. as nearer the truth.[5] Julius Citron,[6] in a recent consideration of the relationship of syphilis to aortic insufficiency, calls attention incidentally to the frequency of lues as a cause of arteriosclerosis, and this same idea permeates all the literature on the subject.

The Wassermann test has been employed by Albert Schütze[7] in cases of aortic aneurysm, only to prove again the existence of a previous luetic infection in almost every case. Confidence in this test is growing rapidly, and it is another instance of a clinical method of precision confirming an opinion which had previously existed for years.

The presence of aneurysm may, at times, establish a syphilitic infection as a certainty. Nonne[8] has recently expressed the belief that reflex pupillary rigidity and failure of pupillary reaction to light and convergence as the only signs of disease are not conclusive of syphilis, since they may be produced by uncomplicated chronic alcoholism. Isolated organic pupillary anomalies with some disease of the aorta, such as aneurysm, however, have in his experience shown themselves only in cases with a syphilitic history, and he shares Strümpell's view that this symptom-complex must be regarded as indicative of syphilis. In the same paper he takes occasion to emphasize the suggestion that the variation in the intensity of the symptoms of diseases of the nervous system caused by syphilis is decidedly less than is usually thought .

Syphilis is likewise, without question, the chief etiological factor of almost all the cases of tabes. Fournier, in 1875, first called attention to the parasyphilitic nature of the disease. He has reported 87 to 93 per cent. of cases as due to syphilitic infection, while Déjerine found 92 to 94 per cent., and Church, out of a series of one hundred cases, found only two which failed to disclose a history of syphilis[9]. W. Erb[10] thinks it probable, though not absolutely proven, that all cases of locomotor ataxia are due to syphilis. Schütze[7] has employed the Wassermann test in cases of tabes also, and has demonstrated the almost constant luetic history in cases of the disease.

In the instances where tabes and abdominal aneurysm are found associated, it is natural to question whether the pains that are experienced may not be partially due to the pressure of the aneurysm on certain nerves. Such a dilation of the aorta, if of sufficient size, may cause pains which are lancinating in character and very similar to those of tabes, particularly if such dilation is located low enough to press upon the lumbar nerves. When high in the abdomen, it impinges upon the solar plexus and splanchnic nerves, causing a more fixed and constant pain in the back. Thus, in a given case, where tabes is known to be present, it is obvious that this symptom of an aortic aneurysm in the abdomen may readily be attributed to the tabetic condition. It is possible, indeed, that such was true in the patient described below. The masking of tabetic pains by the pains of an aortic aneurysm known to exist in the abdomen, could, it seems, just as easily occur.

When we remember, then, that syphilis is an important cause of both aortic aneurysm and tabes dorsalis, and when it is observed that they frequently occur together, the question suggested by Lesser not inaptly arises whether there may be in tabes dorsalis an etilogical factor in the production of aneurysms of the aorta. We are inclined to believe that this association which, as has been shown, not infrequently exists, is merely accidental, and that each, in such cases, is a parasyphilitic condition independent of the other. Gowers[11] considers that valvular heart disease complicating tabes is merely a coincidence of cause—both being of syphilitic origin—and the importance of this view in supporting the contention that the same relation exists between aneurysms and tabes is apparent. The researches of Heitz and Norero[2], briefly referred to above, in which they made a study of eight cases of tabes with more or less ataxia, in order to determine the modifications in the arterial pressure under the influence of gastric crises and lightning pains, do not militate against this view. It is true that during the crises there is usually a vasoconstricting spasm, except in advanced cases in which the reflex phenomena are less active. The peripheral lightning pains were found to determine hypertension, and the inhalation of amyl nitrite is usually followed by the relief of pain at once—both abdominal and peripheral—and this relief, as a rule, proves permanent even after the blood pressure has returned to its normal level. Röhmer[12] has recently attempted to demonstrate that subcutaneous injections of suprarenal extract have a curative effect on the pains of tabes. This, if true, is obviously contradictory to the results of Heitz and Norero, which showed the palliative effect of a vasodilator on the same condition. Notwithstanding these facts and that the pulse was found to be accelerated during gastric crises, the experiences related seem to show that the cardiovascular system does not suffer from the repetition of the tabetic pains as much as one might suppose, and while the increase in intraarterial pressure and pulse rate associated with them might be regarded as a factor favoring the production of aneurysm, yet these changes are not sufficiently marked to carry with them any appreciable weight in the production of these aortic conditions. Moreover, we cannot lose sight of the cases of aortic aneurysm which occur in patients with a history of syphilis where tabes never developed; nor, on the other hand, of the many instances of tabes of syphilitic origin which, while the usual amount of arteriofibrosis may occur, yet there is not the formation of aneurysm. Whether the toxine is unique or more virulent, or the resistance of the patient less, in the cases where the two conditions are associated, must, to a certain extent, for the present at least, remain a mooted question, but we believe the better view supports the opinion of Debove[4] that the coexistence of the two conditions is only a coincidence and entirely independent of the virulency of the toxine, the resistance of the patient or any other factor.

For the privilege of reporting the following case, I am indebted to Dr. Spiller, the patient having been in his service in the Philadelphia Hospital:

J. P., age 39, admitted January 28, 1908, complaining of nausea, vomiting, incontinence of urine and feces, pains in the lower limbs, and loss of power in the left lower limb. His family history was negative. His occupation was that of a huckster. He was a widower with no children and his wife had had one miscarriage. He had gonorrhea several times and a hard chancre at the age of seventeen. He used alcohol in large quantities and indulged in great sexual excesses. With the exception of the present trouble he was never seriously ill.

The present illness began seven years ago with loss of control of the bladder and rectum, and with shooting pains in the legs. The power of walking gradually became worse so that his gait was very ataxic. The shooting pains became intense. The week previous to his admission he was not able to move his left lower limb. He had considerable vertigo and also cough and expectoration.

Examination: Pupils irregularly oval, right reacting feebly to light and the left not at all. Both reacted well to accommodation and convergence.

No ocular palsies, ptosis or nystagmus. Upper limbs showed marked ataxia. Grasp equal on both sides. Biceps and triceps tendon reflexes absent. Lower limbs markedly edematous, and showed some muscular atrophy. Power to move left decidedly less than right. There was resistance to passive movement on each side. Heel to knee test showed marked incoördination on both sides. Patellar and Achilles tendon reflexes absent on both sides. No plantar reflex or ankle clonus. There was inability to walk or stand. Sensation impaired over entire body—practically absent in lower limbs. Sense of position was greatly lessened. Mentality was good and speech was normal.

Eye examination showed vision to be 4/5 in each eye. Media clear, discs pale only in papillomacular bundle. Vessels normal in size.

He gradually grew weaker, the cardiac condition increased in severity, and he finally died on March 14, 1908.

The autopsy findings were, in addition to chronic tuberculosis of both pulmonary apices, chronic fibrous endocarditis of mitral and aortic valves. tuberculous ulceration of small intestine, chronic parenchymatous nephritis, a large retroperitoneal mass which was found to be a false aneurysm. Beginning at the diaphragm, the aorta widened out into an irregular sac which was connected with the lumen of the aorta by an opening 4 cm. in diameter. The edges of the opening and adjacent tissue were made up of tissue similar to that of the aortic intima. The bodies of the last dorsal and first lumbar vertebræ were much eroded. Over the lower vertebræ lay a firm, yellowish-white membrane. At the margins of that portion of the sac whose walls resembled the intima of the aorta, and whose boundaries were difficult to determine, began a white stratified clot which lined the false aneurysm. Inside of this was a currant-jelly clot. The original sac measured 12 cm. in diameter while the false sac extended on either side from the diaphragm to the iliac region.

Gross examination of the cord showed a diminution in its size with a thickened and slightly opaque pia. The posterior columns were visible as a gray band throughout the whole length of the cord. On section, the contraction of the posterior columns was conspicuous, and a gray translucent appearance was evident. The posterior nerve roots shared in the atrophic process, and were small and gray. Minute examination showed intense degeneration and sclerosis of the posterior columns and roots with marked round-cell infiltration of the pia.

With the exception of a somewhat thickened pia, infiltrated with round cells, there was nothing abnormal found in the brain.

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332 South Fifteenth Street.

FLACCID PARALYSIS OR EXTREME HYPOTONIA OF THE MUSCLES WHICH POISE THE HEAD, AND OTHER SYMPTOMS INDICATIVE OF A CEREBELLAR TUMOR.

By Charles K. Mills, M.D.

(From the Department of Neurology of the University of Pennsylvania.)

Although a necropsy was not obtained in the following case, in spite of earnest efforts to this end, and therefore the nature and the position of the lesion were not determined, the symptoms presented were in one or two respects so unusual that it has seemed worth while to put the case on record.

The history of the case and the focal symptoms pointed to a cerebellar lesion; the general symptoms—headache, nausea or vomiting, vertigo, and papilledema—to an intracranial tumor. The symptoms which were of course most indicative of cerebellar tumor were the vertiginous seizures which were accompanied by vomiting, the ataxia, and the tonelessness or paresis of the muscles of the neck, back and trunk.

The inability to support and poise the head was a striking symptom, and was not one which came on in the last stages of the disease. The mother reported that she had first observed the tendency of the head to fall to one side or forward several months before the death of the patient. The inability to hold the head erect was almost absolute, amounting to an extreme hypotonia or flaccid paralysis. I have never seen a case in which this loss of power was so marked.

Child eight years old with general symptoms of brain tumor—seizures with vertigo, vomiting and falling—development of tendency to gradual drooping forward of the head—dizziness caused by efforts to hold up the head—weakness of right leg and arm—examination showed inability to support and poise the head—trunk movements generally weak or unskilful—recurring spasticity in the right lower extremity—deep and superficial reflexes exaggerated, especially on the right.

The patient, M. M., eight years old, was admitted to the Philadelphia General Hospital, November 4, 1908. The mother of the patient had two other children older than the patient and three younger; she lost two children with bronchitis. The father was a miner and in good health. The mother had always been in good health. According to the statements of the mother, the child was well until one year before admission. She was active physically and mentally, had been going to school, and had no ailment of any sort. She had had measles a year or two before this time, but had no other illness with the exception of occasional colds. She had a fall from a chair when she was a child, but was not rendered unconscious by the fall nor had any trouble afterwards.

One year before admission the patient went to school as well as ever and at two o'clock in the afternoon was brought home by two girls who said that she had fainted in school. It was afterwards learned that the child had vomited and fallen on the floor in a faint in the school room. When asked as to whether or not she was unconscious the mother said that she seemed to know that she had fallen from her chair, but could not get up without assistance. When she reached home she was pale and very cold. She lay down and slept after reaching her home, and at the end of two hours got up apparently as well as ever. Two weeks after this she had another attack similar to the first. This one occurred at home. The patient first reeled around and around and then fell to the floor in a faint. She had had four of these serious spells since the first one. Besides these attacks the patient had another form of seizure in which she became sick at her stomach, vomited and complained of dizziness and sometimes, although not always of headache.

About six months before the admission of the child to the hospital her mother noticed for the first time that she held her head in a curious way, usually drooping it towards her chest. At first she thought that she tended to carry her head to the right side. She gradually got so that she could not hold her head up when sitting or standing, saying if she attempted to do so she became very dizzy.

Three months before admission to the hospital the patient began to drag her right foot a little and also to use her right arm not so well as she used the left. She also at this time, began to take on a wabbling station and walk. She did not seem to be able to control her legs fully, the right being weaker than the left.

On her admission the child complained of pain in both the frontal and occipital regions, and suffered from dizziness. She lay upon the right side, the lower extremity drawn upward, and the right hand pressing upon the forehead. She had the eyes partly closed, with occasional movements upward of the lids and eyeballs. Her speech was slow and she was stuporous and apparently sleepy. She was aroused with difficulty. When asked as to the duration of her illness she gave uncertain answers, but complained of pain in her head, vomiting and dizziness. She yawned frequently. She lay in rather apathetic state, occasionally moaning. Efforts to rouse her were not entirely successful, but to a certain extent she recognized her surroundings, but she did not do what she was told. At times she would try to do what she was told, and then would say distinctly, "I can't do it."

Physical examination as to heart, lungs and abdomen was negative with the exception that at times she had an intermittent pulse. Her bowels and bladder were occasionally incontinent.

The patient was apparently unable to sit alone for any length of time or to stand by herself. In making the effort it seemed that the trunk muscles would not act properly in the sitting position and in standing the head would be held for a moment in the line of the trunk and then droop to one or the other side, and losing the center of gravity the child would topple and fall over. This inability to support the head was evident whether she stood or sat; the head would fall in any direction, but never with any constancy in any one direction. The head could not be held in an erect position, but without support would fall backward, forward or sideways. During the examination the neck muscles were not spastic; they were paretic, flaccid or toneless. She did not seem to use them. Resting on the pillow she was able only in a feeble manner to rotate her head from one side to the other.

The child had great difficulty in chewing and swallowing. In fact the muscles for mastication were used only to a slight extent. The lower jaw hung downward, and there seemed to be some weakness of both sides of the lower half of the face. The patient could wrinkle the brows and close the eyes tightly.

The tongue was protruded in the median line with very slight tremor.

The upper extremities showed, in common, a peculiar flail-like condition with motion only as the result of what appeared to be extreme effort. The right upper extremity, however, at times seemed to be slightly spastic. The reflexes were slightly increased, more on the right than on the left. There was, apparently, no atrophy in either of the upper extremities.

During the examination of the lower extremities the patient moved the legs under the bed clothing, but endeavor to bring out resistance in them was without result. The right leg, at times, seemed to show some temporary spasticity. The deep reflexes were all increased. Foot clonus on the right side was marked and persistent; on the left it was present, but rapidly exhausted. Holding the feet at the instep and stimulating the plantar surface on the right, a typical Babinski was obtained; on the left, under the same conditions, hyperextension of the great toe was obtained, and also the tarsal movement. The von Bechterew tarsal reflex was not obtained.

Examination for sensation was very unsatisfactory. The patient seemed to respond to all forms of stimulation, these responses being decided by the frown on the child's face when the irritation was offered.

Testing the patient for general vision, she recognized a watch and a twenty-five cent piece, but she seemed slow in apprehending what was asked of her or what she was told to do. The pupils were about equal, of moderate size, each reacted promptly to light and in accommodation. The eyelids were not completely approximated when the child was resting or was sleeping.

The eye examination showed, in the right eye, a small corneal scar at the inner edge. The disc in this eye was swollen, the temporal edge being visible. The surface of the disc was plus three diopters. The arteries were about normal in size; the veins were enormously swollen; particularly the upper and lower temporal veins were very tortuous. Near the disc at the inner side was a linear hemorrhage. The left eye showed a slightly hazy cornea. The disc was swollen, and the surface of the disc was plus two and one half diopters. The arteries were normal, but the veins were enormously swollen as on the right side. Both discs showed marked pallor in the deep layers, indicating the beginning of atrophy. It was impossible to take either the vision or the fields. There was no strabismus, but rotation of the eye was not possible.

After the examination the patient was weak and exhausted. On November 8, 1908, four days after admission to the hospital, the child became cyanosed, pulse almost imperceptible, and death followed. No necropsy could be obtained in this case.

It is well known that in lesions of the cerebellum, especially tumor, the head sometimes tends to one side, according to most of those who have observed the phenomenon, away from the side of the lesion. Owing to the flaccidity or tonelessness of the muscles of the trunk and neck, the head and body may assume all sorts of abnormal positions in the effort of the patient to maintain balance. As stated at the beginning of this report, however, I have never seen a case in which the inability to maintain the head in a fixed position was so marked as in the one here reported. The loss of control over the muscles of the trunk was also more marked than in almost any case which has come under my observation.

THE DIFFERENTIAL DIAGNOSIS OF GRAVE HYSTERIA AND ORGANIC DISEASE OF THE BRAIN AND SPINAL CORD, ESPECIALLY DISEASE OF THE PARIETAL LOBE[1]

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(From the Department of Neurology of the University of Pennsylvania.)

Several reasons seem to make it desirable to present this case to the society for investigation and opinion. Owing to the peculiar mental state of the patient and the manner in which his symptoms improved after admission, the question of diagnosis as between an organic and a grave hysterical affection is worthy of some consideration. The clinical phenomena in this case, at least most of them, are, I believe, to be regarded as of organic origin, in spite of the great improvement which has taken place in a period of a few weeks.

Much interest has been centered upon the diagnostic value of two of the symptoms presented by this patient, namely the foot clonus and the Babinski sign. My own opinion, long since expressed at a meeting of this society and before the meeting referred to[2] is that persistent foot clonus is almost invariably a sign of organic disease. It usually means destruction or interruption of the cortico-spinal fibers, although disease of the motor cortex may also give this manifestation. Now and then a case is seen which would seem to throw doubt upon this position, but always, I believe, careful investigation will show that it is tenable, in other words that the cases in which foot clonus is present, and which are supposed to be hysterical, have some organic basis. Of course it is well known that an abortive or spurious form of foot clonus may be seen in grave hysteria, and even in severe neurasthenia, but this is very different from the persistent trepidation which is observed in organic cases, and which is quite marked in the patient exhibited to the society.

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[1] Read at the meeting of the Philadelphia Neurological Society, held December 18, 1908.

[2] Proceedings of the Philadelphia Neurological Society, Journal of Nervous and Mental Disease, July, 1897, Vol. XXIV, No. 7.

The Babinski sign when typical is also, as Babinski himself avers, always an indication of organic disease, and especially of disease of the pyramidal tract. This view I continue to hold, notwithstanding the recent paper of Van Gehuchten[3] who has been converted to the opinion that in rare cases the Babinski sign may be present in hysteria, this contention being founded upon the report of a case studied by him in which both foot clonus and the Babinski sign were present and both disappeared in a few days after their demonstration. One case, however well studied and however striking, is not sufficient to contravene the testimony of scores of other well-studied cases. Both foot clonus and the Babinski sign are present in the patient here presented. The Babinski sign is indeed manifest on both sides, although more demonstrable on the left. The inference from a study of these phenomena is therefore in favor of organic disease.

Hypesthesia is of course frequently present in functional disease, but it is equally true that it is sometimes the sign of an organic affection and especially of disease of the parietal lobe.

Whatever may have been the experience of others, I have yet to see a case of true astereognosis of hysterical origin, and I am always doubtful as to the functional nature of cases in which loss of the sense of position and ataxia of the upper extremity of the type presented by this patient are present. A pseudo-ataxia, like a pseudo-foot-clonus, and a pseudo-Babinski sign, are all seen in hysteria but can be readily differentiated from the corresponding symptoms with an organic basis.

It is worthy of consideration in discussing the question of hysteria, to remember that the hysterical imitations of organic disease are usually those which are concerned with what might be called elemental nervous phenomena, such phenomena as impairment of cutaneous sensibility, of motility, and lowering of the functions of the special senses. Stereognosis is a more highly evolved cerebral function than cutaneous sensibility, and its loss, like true word deafness and word blindness or letter blindness, never occurs in pure hysteria. This is an interesting fact, and one which sheds some light upon the nature of hysterical phenomena, but this phase of the subject I shall take up later in a paper to be read before one of our national associations.

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[3] Van Gehuchten, A., Le Nevraxe, Recueil de nevrologie normale et pathologique, Vol. VIII, Louvain, 1906.

I might say in passing that I have never seen typical hemianopsia in a case of grave hysteria, although I have of course seen many instances of peculiar alterations and contractions in the visual field. Here again the explanation is to be found in a consideration of the real nature of hysterical phenomena. Complete loss of one half of the visual field produced by organic lesion of the optic radiations or by lesions in other positions is a very different phenomenon from the visual fields often presented by hysterical patients.

The diagnosis in the present case is somewhat doubtful, but the probabilities are that it is one of cerebro-spinal syphilis, possibly general paresis in an early stage. It is of course a fact too well known to be commented upon here, that both in cerebral syphilis and in general paresis the patients are the subjects of apoplectiform attacks, such as this man suffered from and such as he clearly describes. It will be seen from a study of his symptoms that he has some evidence of disease in both lower extremities, where the reflexes are exaggerated and where the gait tends to assume a spastic form, although his most pronounced symptoms are unilateral and left-sided. The case may be one in which, in association with some spinal disorder like a not much advanced meningomyelitis, more pronounced cerebral lesions are present in the parietal and frontal lobes.

This case is particularly instructive if the views advanced in its presentation are correct, in the differentiation of hysteria from lesions of the parietal lobe. He has the typical parietal lobe symptom complex, plus other manifestations. These parietal symptoms are the hypesthesia, impairment of the muscular sense, the ataxia, and the astereognosis. As I have tried to indicate, some at least of these symptoms are never present in hysteria, however grave. The foot clonus and Babinski sign are to be explained not by disease of the parietal lobe but by involvement, either in the cord or in the brain, of the corticospinal systems of fibers.

J. D., male, forty-seven years old, was admitted to the Philadelphia General Hospital, November 22, 1908. He showed at the time of his admission much impairment of power in the use of his left arm and leg, and a psychic state, which in general terms might be described as one of mild optimism or exaltation.

His mother died of consumption; otherwise his family history was negative.

The patient was married twenty years before the date of admission. His wife had no children, but had one miscarriage. He had used tobacco to excess, used tea and coffee moderately, but did not use alcohol. He had a chancre when nineteen or twenty years old, but never had a rash. He took specific treatment for two years.

During the year previous to his admission he stated that he had been treated for nervous prostration which was brought on by care and worry. He said that his most important symptoms had been in his lower extremities, his right leg making what he called a "sloppy movement." He was well enough to be able to be up and around until two days prior to his admission to the hospital.

At about eight o'clock in the evening of this day his right eye began to smart, and he felt a numb or tingling sensation creeping from the right side of his forehead to the left. Headache was present to a slight degree and he felt as though he were going around. He then walked up one flight of stairs, lit the gas in his room, sat on the bed and suddenly became utterly helpless. The left side of his body would not work at all. He could not stand or walk, so he was put to bed and medical aid was summoned. The left leg and arm lost "their intelligence" as the patient expressed it, while the attending physician told him that he had a stroke and that the left leg and arm were somewhat paralyzed.

Consciousness was never lost, although the patient said that he might have been dazed for a few moments. He said that his speech was affected in that he was not able to talk as fast as he had formerly, while both his vision and hearing were diminished. The left half of his tongue felt thick and clumsy.

On admission to the hospital the patient complained of no headache or pain, but had vertigo when he rose to a sitting position, which he was unable to do without aid. His mind was in a peculiar state; there was a certain sense of well-being present, and at times he appeared to be what one would call "fresh" or flippant.

He was a fairly well developed and well nourished man.

Examination showed that the conjunctivæ were normal: the pupils were slightly dilated, but equal, regular and responded both to light and in accommodation; extraocular movements were normal and no nystagmus was present. The face was slightly congested. The masseters contracted equally on both sides. The mouth was opened in a straight line. He wrinkled the forehead and closed the eyelids well on both sides. He showed his teeth and drew up the angles of his mouth fairly well on both sides, but the movement on the left was slower and not so well performed as that on the right side. The tongue was clean and moist; it was protruded in the median line and was freely movable. No tremors of the lips or tongue were noticed. Swallowing and mastication were normally performed. His speech was not apparently affected.

The pulse was about normal in rate, regular, of fair volume and tension; the vessels showed some sclerosis. The chest was well formed; expansion was good, but somewhat greater on the right than on the left side. The heart and lungs were normal. The abdomen showed no abnormality. The musculature of the upper extremities was good; no spasticity was present on either side.

All movements of the upper extremities could be performed on both sides, but on the left they were somewhat slower and lacked certainty as compared with the right. The left side was slightly weaker than the right as demonstrated by movements against resistance, while the grip was also somewhat weaker on the left, although fairly strong on both sides.

Marked ataxia was present on the left side as was demonstrated by the finger-to-nose test and even better by the finger-to-finger test. The right side was not ataxic. No tremors were noticeable on either side.

In the lower extremities the musculature was good. Very slight spasticity was exhibited in both limbs. All movements could be performed on both sides, but the left side was somewhat weaker than the right. The left side showed some ataxia, but none was evident on the right. The station and gait were not tested at first.

The patient had normal control of both the bladder and rectum.

Studying the cutaneous and deep reflexes resulted as follows: The conjunctivæ and palatal reflexes were present on both sides; the abdominal, cremasteric and plantar reflexes were normal; the biceps, triceps and patellar tendon reflexes were increased on both sides; patellar clonus was present on the left side; the Achilles tendon reflex was increased on both sides; persistent foot clonus and the Babinski sign were present on both sides. Hyperextension of the great toe could also be developed by the methods of Oppenheim and Gordon.

Examining the patient for sensation, it was found that tactile sensation was diminished very moderately on the left side of the body, principally in the extremities. This was also diminished on the left side of the face below the level of the eyes, while the trunk and abdomen were little, if any, involved. In the extremities this diminution became more marked as the hand and foot were approached. Pain and temperature senses were diminished over the same area as was tactile sense. The diminution of the temperature sense was not so marked as that of the other senses. The patient could not tell difference in pressure as well on the left as on the right side of the body. His sense of locality was fairly good on both sides. The sense of position was diminished on the left side. The patient could not, with his eyes closed, exactly place the right limb in the same position in which the left limb had been placed. He was unable to tell accurately the position in which his left leg or arm was.

Stereognosis was normal on the right; on the left side astereognosis was present.

The patient's central vision was somewhat diminished in both eyes; peripheral or field vision was about normal for both eyes.

An ophthalmic examination was made by Dr. G. E. deSchweinitz on December 2, 1908, who reported as follows: Physiological cupping was marked in the left eye; the eye grounds were normal.

Hearing was diminished on both sides, more on the left than on the right. The tick of a watch could not be heard when placed in front of the patient's ears or upon the mastoid processes.

Olfaction was normal. Taste was normal on both sides of the tongue.

The man's memory was good for both past and recent events. Intelligence was good. He had no delusions or hallucinations.

The following notes were made on December 6, 1908, fourteen days after his admission to the hospital.

The patient was still in bed, but was able to rise to a sitting posture with little or no discomfort. The strength on the two sides of the body was nearly or quite equal. The left angle of the mouth was drawn up as well as the right.

The ataxia had diminished somewhat on the left side of the body but was still easily apparent. Tactile, pain, temperature and pressure sensations had improved on the left side, but were not normal. The sense of position had also somewhat improved on the left side. Astereognosis was present on the left side, but now was rather a hypostereognosis. The reflexes were about the same as at previous examination.

On December 11, 1908, the patient was up and walked around for a few minutes, but needed assistance. His station was fairly good, with eyes opened or closed. His gait was rather spastic, some ataxia being apparent in the left leg, which became more marked when the eyes were closed.

On December 20, 1908, the patient walked without assistance. His gait was distinctly less spastic and awkward than at the time of the previous record. Ataxia was not noticed in the left leg. The ataxia had decreased in the left upper extremity, so that at this time it was present but to a slight extent.

Tactile, pain and temperature senses were normal on both sides of the body. The sense of position was markedly improved on the left side.

Ability to recognize objects with the left hand had distinctly improved, but was not normal. Hypostereognosis was present. The reflexes were the same as when admitted.

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STUDIES WITH THE WASSERMANN REACTION.*

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THE part played by syphilis in the production of diseased conditions of the nervous system has been under discussion for many years. If the complement-fixation tests prove unquestionable, these problems will be much simplified. Already the Wassermann reaction for the diagnosis of syphilis has given rise to an enormous amount of work which, despite individual variations and modifications of technique, have been uniformly confirmatory of its value as a means of clinical diagnosis.

In the following report an attempt has been made, while studying those nervous diseases due directly or indirectly to syphilis, to compare the Wassermann reaction, the Noguchi modification (using, howover, the fluid reagents), and the globulin tests. The precipitation tests were included at first, but discarded, our results according entirely with other observers, the largest percentage of positive results occurring in parasyphilitics, and the next highest in nonsyphilitics.

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*The material in this report was taken principally in the Neurological Wards of the Philadelphia Hospital, in the service of Dr. James Hendrie Lloyd, and the cases were collected under his diagnostic guidance. The work was done in the Laboratory of Neuropathology of the University of Pennsylvania with Dr. Spiller.

The technique of the complement deviation tests, Noguchi and Wassermann, have been so often described that we will not repeat them. In every series of tests the quality and strength of all reagents, including a series of antigens, were carefully tested, and dosed by the same normal and syphilitic sera and same complement.

These antigens were alcoholic extract of syphilitic fetal liver, normal liver, guinea-pig heart, guinea-pig liver, lecithin, sodium, taurocholate, sodium glycocholate, and three different mixtures of bile salts and lipoids. These mixtures so far have not given satisfactory results, and are not included in this report. The glycocholate and taurocholate also were unsatisfactory and are omitted. Every test was fully controlled with normal serum, syphilitic serum, and, when possible, by serum of some nonsyphilitic disease.

There was a marked uniformity of result with all four tests. The Noguchi modification was in most cases more sensitive than the Wassermann, this increased delicacy making it give positive results in nonluetic conditions (tuberculous meningitis, one case). The most positive results with the fixation tests occurred in active secondaries, and the faintest in the treated tertiaries. The intensity of the complement fixation tests showed a direct relation to the acuteness of the manifestations and absence or incompleteness of treatment, or length of time since thorough treatment, but showed no relation to severity of the lesion. The

Noguchi butyric acid and the Gay and Fitzgerald euglobulin test varied little in end results. The globulin tests were more often positive in parasyphilitic cases where the deviation tests were less constant. It is of great value, as corroborative evidence in distinguishing between brain tumor and cerebrospinal lues, where the fixation tests were often partial or absent.

The Noguchi butyric acid test, when applied to cerebrospinal fluid, gave a positive result in every luetic case with meningeal involvement, but failed in one case in which there was no involvement of the nervous system but a gumma of the liver, the blood serum of which was faintly positive. The test was positive also in every meningeal disease examined without reference to cause. The euglobulin was negative in nonluetic meningeal conditions, but present in some acute infections, tonsillitis, and pneumonia. Both of these tests fail if serum is bloody or tinged with hemoglobin.

In the Wassermann tests, in which three or four antigens were used, we found that luetic fetal liver, normal liver, guinea-pig heart, and lecithin ran roughly parallel, the titers for the different antigens varied even when the dose had been determined for all antigens with the same normal and syphilitic sera. In five of the primary cases, the reaction was studied with at least four, and whenever possible more antigens, and in these, during a period of twenty weeks, we observed the same general curve. Lecithin and guinea-pig heart being closest in titers to the fetal liver. In these cases the reaction was positive, first with the Noguchi modification, later with the Wassermann, using the luetic liver and lecithin antigens.

In the parasyphilitic disease the titers were less in early than in later stages. In tabes we more often had a positive reaction when there were active manifestations. One case reexamined during a seemingly latent period gave no change in the reaction, while another whose first test was very faintly positive to Noguchi modification, and negative to Wassermann, gave a definite positive to both tests in a second examination following the development of a hemiplegia. The stronger reaction where both serum and cerebrospinal fluid were used was found in the serum to both the Noguchi and the Wassermann techniques, but in cases where the serum alone was tested, the Noguchi technique seemed more delicate, while in cases where the cerebrospinal fluid was examined the best results were with the Wassermann technique.

Concerning the diseases studied little need be said. In the hemiplegic cases, amyotrophic lateral sclerosis, pseudobulbar palsy, epilepsy, and one brain tumor case, a definite history of syphilitic infection was obtained in all but one of the positive reactions. This patient, a hemiplegic, thirty-two years old, markedly improved under specific medication with which he had been treated for a month before the reaction.

A Wassermann reaction means syphilis active somewhere in the body, and the present trouble may be due to an entirely different cause. A positive pneumonia case seen at autopsy had a gumma of the liver. A positive reaction in this case could easily be recorded as a positive pneumonia. It is not improbable that many times a low grade syphilis may be present, thus making a soil suitable for another infection. Winfield, examining twenty-nine children with tuberculous joint disease, found physical signs of hereditary lues in ten cases, Pirquet reaction in twenty-eight of the cases, Noguchi modification in ten cases, a report strongly suggestive of the presence of both infections.

Seven suspicious pregnant women examined by us gave negative results. Five of these were again examined with the children after labor, with consistent results, except that one baby gave a partial response, but so far it has developed no signs of syphilis. Bar and Duanay think there may be some other factor present in the blood of the newborn which modifies the reaction. They found this weak reaction usually present in sera with bile pigment, or in a lactescent serum. With these two other doubtful mothers and their infants were examined, and were both positive.

Bar and Duanay[1] found the reaction frequently negative in infants whose mothers had been thoroughly treated, and at times in infants whose mothers had florid syphilis, but the highest positive results appeared in those cases in which the disease of the mother was latent. A positive test in the mother justifies treatment of mother and child, but the best prospect of success in treatment lies in energetic mercurialization before and during pregnancy. In the case of a child of a Wassermann-positive mother, which itself gives at birth a negative result, the reaction may later become positive, suggesting the possibility of infection during delivery.

Baisch[2] found in mothers of typical syphilitic children only about 75 per cent. which reacted with the Wassermann test, although he thinks all the mothers were infected. On the other hand, positively luetic mothers may give birth to infants which give negative reactions and remain free from signs of disease. In our cases, we examined the milk in six of these cases, and obtained a positive reaction in the two luetic cases. The reaction was faint in both, and so faint in one by the Wassermann that, except in the light of other findings, it would have been counted negative. In both these cases specific medication was carried out in child and mother, and, except for an eczematous rash on the genitalia of one child, all have been perfectly well during six months of observation.

In five cases of primary, nine of secondary, seven of tertiary, four of cerebrospinal lues, and four hereditary cases, we were able to make frequent examinations of the serum while under treatment for a period of six months.

In the primary cases the reaction was negative until the third week. One gave a sluggish negative to the Noguchi modification in the second week, but was distinctly positive at the third week to the Wassermann and Noguchi modification. These early cases and those latent cases giving negative results may be due to a state of equilibrium existing between spirochete and tissue of host. The variations in the number of positive results in primary cases of different observers are probably due to differences in the duration of the disease when blood was taken. The length of this negative period must depend on individual reaction to toxin, and to the malignancy of the infecting organism. Blumenthal and Roscher[3] found the reaction negative until the sixth to the eighth week. Levaditi, Laroche, and Yamanuchi[4] found it as early as the fifteenth day, and Lesser[5] reports one case where the reaction appeared before the initial lesion.

After the appearance of positive inhibition in our cases, the reaction gradually increased in intensity until modified by treatment. This modification is not always distinct, but follows a definite curve, which does not correspond with, or run parallel to the clinical signs, and varies markedly in different individuals. The Noguchi modification followed the same general curve. The change in intensity of reaction was in every case gradual, and in one case a sudden increase occurred in reaction with no increase in clinical manifestation, and in another (L. M.) there was increase in symptoms with no increase in reaction. In these cases the treatment was in every case inunctions of mercury, and when, despite treatment, the reaction continued to increase in intensity, injections twice weekly of calomel 0.1 gm. were added, with an immediate change in the reaction. In every case when the reaction first became negative, it was repeated, using (1) the nonheated serum, (2) serum to which 1/3200 of N (HCl) was added. In one instance each the reaction became positive again with this manipulation. This never occurred with a normal serum.

In some cases of disease the reaction becomes negative quickly, in others even intense treatment fails to secure a total negative reaction. In one case the lesions yielded very promptly, but at the last examination (4 months) the reaction was still rather strongly positive. In some cases the reaction will become negative without further treatment, as it did in one case of ours. In two of our cases, treatment was stopped by patients at healing of the lesion, and recurrance has already taken place in one, and the reaction is present in both. Blascho[6] thinks that treatment should continue until hemolysis occurs promptly but continued as long as it is sluggish.

Pürckhauer[7] found 35 per cent. of primary and secondary cases became negative under treatment, and thinks that a positive reaction should always be considered indication for further treatment, continuation of the reaction indicating a stubborn case, and a negative reaction should mean response to treatment; and when, after repeated tests at intervals, the reaction remains negative, the patient is cured, or is not at present suffering from a manifestation of the disease. In the primary cases all but one became entirely negative—this man was alcoholic. Two developed profuse general rashes. Of the secondaries six became negative, and the others faint. Of the tertiaries three are practically negative (sluggish), two faint, and two have moderate reactions. Of the cerebrospinal lues all are fainter than at the beginning, but are positive. The same is true of hereditary cases. All the negative tertiary cases we report had had long series of treatment. Two negative secondaries after suspension of treatment became positive again. In three cases in which iodides alone had been given, there was in two no change in the reaction, and in one a slight increase. Blascho succeeded in changing the reaction with iodides. In one case of overmercurialization atoxyl was substituted. The patient had at that time a weak specific reaction, and, after three weeks, the reaction remained practically the same. There was a very slight increase in titers. Alcohol, Blascho[8] thinks, interferes with the change in reaction, and in one of our primary cases this was true. He found that the reaction faded gradually until it became negative, then gradually increased until it was positive again, and treatment was again instituted with a like result. The length of time the reaction persists after subsidence of active symptoms varies greatly. Fleischman found it in some six to nine months, in others, usually severe cases, three to four years. In one case of cerebrospinal lues, there had been no manifestation of disease for three years, and partial reaction was present. In another case it was negative seven months after trouble, at nine months was still gone, and at one year was again present. At this time the man had a severe attack of gastroenteritis. In this case it looked as if a new infection had aroused this entirely quiescent syphilis to activity. This return of reaction may occur without clinical signs, and did in this case. The man was, however, put on injections and inunctions immediately upon discovery, and reaction is again negative. Hoehme found that the least change in reaction occurred under treatment by mouth. Thirty-three per cent. changed with inunctions, and the most definite changes occurred with the intramuscular injections. In our experience the most pronounced change in reaction was in the use of two avenues of entrance, preferably inunctions and injections. In early cases the change was more marked than in the later phases of disease. In the later stages, to secure a negative reaction, the treatment must be more vigorous than at present used. In hereditary cases Lesser, Blascho, and others have found no change in the reaction with any forms of treatment used.

The Wassermann reaction does give strong positive evidence as to the presence or absence of active syphilis. As a rule the diagnosis of lues is not attended with much difficulty, but even in the more open cutaneous manifestations doubtful cases occur where it is of great importance. It is necessary to remember that the reaction gives evidence of syphilis in the body, but not the location of the lesion. A man may have glioma, etc., and also syphilis. In using the test to diagnose obscure conditions, the corroboration of the other tests is of very distinct value, especially where the reaction is partial. In cases with no definite history or distinctive clinical signs a positive diagnosis should be given only when there is little or no hemolysis, and there is a marked sedimentation on standing. If the effort, however, is to exclude syphilis or note the effect of treatment, condition of latency, or cure, a faint reaction should be reported as positive, and should be followed by treatment.

As a clinical test, its greatest importance seems to be as an index to treatment, the serum of luetics being examined in periods of health and in neurasthenic states, and during the acute infections for reappearance of antibodies, and the patient being treated before later serious manifestations appear. The small series in which we blocked out the curve of the reaction under treatment seems to point to need of earlier mercurial treatment, and of more intensive treatment from the first. In obstetrics its value is of immense importance to the child. Pregnant women, if examined and giving a positive reaction, may, if treated carefully, give birth to healthy children, or at least children better equipped for the struggle with life. In the diagnosis of paresis and tabes the complement test seems only to verify the finding of clinical history and cytodiagnosis of cerebrospinal fluid. About 90 per cent. of paretics give reaction, and about 70 per cent. of tabetics.

In the study of the etiological part played in diseases of the nervous system, the reaction was of great importance, especially in multiple sclerosis, brain tumor, hemiplegias, epilepsies, and purely luetic troubles. The Wassermann reaction will probably clear up some old problems, and will undoubtedly open up new fields for research.

Excluding leprosy, frambesia, and yaws, we think that a great deal of value is to be placed upon a positive reaction. A negative result is suggestive evidence of the absence of syphilis, but sometimes occurs when active syphilis is present. This might be explained by the fact that the ability of the body to react and form antibodies is exhausted, for it was usually in the old cases of known syphilis that the reaction failed with us.

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THE DIFFERENTIATION BETWEEN DEMENTIA PRÆCOX AND DEPRESSIVE STATES BY SO-CALLED BIOLOGIC BLOOD-TESTS*

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While the Wassermann reaction is of greatest value in differentiating between syphilis and non-syphilitic diseases of the nervous system, it does not permit of further differentiation between so-called functional nervous diseases.

Recently Much published a description of a method by which, he believed, he could differentiate dementia præcox and manic-depressive disease from other diseases. The method is based on the inhibiting action of blood-serum in cobra hemolysis. His results have not been confirmed by others.

In the following we wish briefly to state that we seem to have been able to differentiate by relatively simple methods between dementia præcox on the one hand and melancholia on the other.

Our method is based on the determination of the fixation of complement of guinea-pig serum by the serum of the patient. We found that in melancholia the amount of complement absorbed is very much larger than in all other diseases that we have encountered, and also much larger than in dementia præcox, in which disease it is approximately normal.

We have tested 67 cases of various mental diseases, 37 cases of melancholia half of which were of the manic-depressive type, and the others involutional. So far our test has been decisive and characteristic in all cases examined. We shall continue the examination for possible exceptions.

We also wish to state that it is possible to differentiate more clearly between mental diseases by using very carefully standardized factors in Much's method. In our cases this reaction always occurred in dementia præcox and almost never in melancholia, when all the factors were standardized.

In conclusion we wish to say that our results distinctly indicate that in so-called functional mental diseases, chemical changes in the blood take place, and can be discovered by so-called biologic methods.

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A REVIEW OF SERUM REACTIONS IN CASES OF NERVOUS AND MENTAL DISEASES[1]

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Pathogenic conditions reflect themselves in the blood, and the nervous system is often the first to suffer from the dyscrasia, and yet the many years of careful and systematic morphological study of the blood in neurological and psychiatric practice have yielded little of diagnostic importance. A picture changing from normal to varying degrees of anemia, generally of a chlorotic type, was found. Many interesting single facts were added, but the blood deterioration could only rarely be regarded as a factor in the insanity or nervous phenomena. Every blood test described has been applied to these conditions, but until the advent of the Wassermann[2] reaction, in 1906, no evidence of etiological value was derived from the study of the blood, except in rare instances. With the complement fixation test, however, a new and large field was opened up, not only to decide upon the syphilitic genesis of diseases, but to watch the effect of treatment, and to clear up the causes of some of the obscure conditions occurring in the nervous system. The importance of this reaction in all departments of medicine increases as the better command of the complicated

[1] Read at the twenty-sixth annual meeting of the American Neurological Association held in Washington, May 2, 3 and 4, 1910.

[2] Wassermann, Neisser und Brück; Deutsch. med. Wchnschr., 1906, XIX, 745.

technique adds to its specificity. In neurology its value is very great in distinguishing between syphilitic diseases of the central nervous system and central gliosis, tumors, multiple sclerosis, etc.; but its importance is not yet fully appreciated by the general profession.

In this paper we wish to report the result of two years' work with the Wassermann reaction, its modifications and simplifications, with a parallel study of some other changes found in the blood of mental and nervous diseases. Practically all forms of mental and nervous trouble were subjected to Wassermann's, Noguchi's, Weil's and Much's tests. The conclusions in this paper are founded upon examinations made in 1710 cases of nervous and mental diseases. Our conclusions in the other tests, *i. e.*, Fornet and Schereschewsky[3] precipitations, globulins, etc., are based on at least three hundred cases each.

Fornet and Schereschewsky's test shows the relation between the precipitin reactions and the binding of complement. In those cases where no visible precipitate occurs on the contact of precipitinogen and its specific precipitin, a binding of complement is still demonstrable. As a diagnostic aid, the Wassermann or the Noguchi reaction is the most reliable and delicate test for syphilis. The precipitation tests with distilled water, dilute alcohol, taurin, sodium glycocholate, sodium taurocholate, lecithin, etc., while of some scientific interest, are, we think, too unreliable for diagnosis. The percentage of positive results are highest in parasyphilitic diseases (92 per cent.), and, in our experience, next highest in non-specific disease (38 per cent.).

Of much more value is the butyric acid test devised by Noguchi.[4] This test demonstrates the almost constant increase in the globulin of the blood or cerebrospinal fluid in luetic infections—an increase less definitely shown by Klausner's[5] precipitation test. Noguchi, when studying the globulins of syphilitic sera and cerebrospinal fluid, found that the euglobulin fraction held the binding substance, and would itself give the Wassermann reaction with the same titer as the original serum. Based on this observation, Gay and Fitzgerald[6] devised an euglobulin test.

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[3] Fornet and Schereschewsky; Münch. med. Wchnschr., 1907, LIV, 1471.

[4] Noguchi, Serum Diagnosis of Syphilis, J. B. Lippincott, 1910.

[5] Klausner, Wein. klin. Wochschr., 1908, XXI, 940.

[6] Gay and Fitzgerald, Boston Med. and Surg. Jour., CLX, 1908.

Both Gay's and Noguchi's globulin tests are of confirmative value. The euglobulin test is a little more clearly cut, but possesses otherwise little or no advantage over the globulin test. The increase of globulins has been noted experimentally in the process of immunizing animals to bacteria or organic substances. We obtained positive results in one case of tuberculous meningitis, one of caries of lumbar spine, and in one brain tumor. In the last, however, the Wassermann reaction was positive. Gay and Fitzgerald[6] report cases of positive results in acute infections. These should give rise to no confusion, however, if all factors of the case are taken into account. In the cerebrospinal fluid, any inflammation of the meninges will give positive results.

The globulin increase appears before the Wassermann reaction and remains longer in cases under treatment. In varying degrees, it constantly accompanies primary, secondary and tertiary syphilis, tabes and paresis. In paresis the test is strongly positive in the blood and cerebrospinal fluid. In cerebrospinal lues and tabes, it is a much more constant symptom than the complement fixation test, which is often weak or absent. The non-specificity of the test detracts from its diagnostic importance, but not from its corroborative value. In our experience the absence of the butyric acid reaction is more proof of the absence of syphilis than the absence of the Wassermann reaction. The necessity of performing this globulin test appears in those cases with a luetic history, which are being watched for effect under treatment. Every case in which treatment was stopped while this increase in globulin persisted had a recurrence of Wassermann reaction in a short time. As an illustration: E. M. received a severe injury to his head and back, was unconscious four hours, then gradually recovered and went back to his work. Seven months after his injury, he developed a typical spastic paralysis. When seen by us eleven months later he denied lues and any specific medication and the Wassermann and the Noguchi reactions were negative, but the globulin was faintly positive. He was put on tonic treatment, and, at the end of two months, the globulin test was strongly positive, Noguchi and Wassermann reactions were present. In this case, probably, the injury lowered the resistance, so that a weak latent lues became active. The absence of the first reaction was due to treatment, the nature of which had not been understood by patient.

Another corroborative test for syphilis is that proposed by Weil,[7] depending upon the alterations in the erythrocytes in response to the syphilis toxin. He found that the red cells, after infection with syphilis, exhibited first an increased fragility toward some hemolysins, especially toward cobra venom, and that later they acquired a marked resistance. The increased resistance appears later than the Wassermann reaction, and remains longer than any test in treated cases. In our study we obtained positive results in 94 per cent. of the cases of frank syphilis, one case of paralysis agitans, and in only 20 per cent. of cases of syphilis of the nervous system, including tabes and general paralysis. The test depends upon the changes in the lecithin content of the cell, of which cobra venom is an indicator. Weil suggested that the negative reactions in cerebrospinal lues were due to the fact that the virus of syphilis will spare the lipoids of blood cell when it attacks those of the nervous system. When the amount of lecithin is decreased, the Wassermann reaction and Weil's test are positive. A negative outcome of a fixation test in a case of positive lues (errors in technique excluded) may be due to an elevation of the lecithin content. Quarrelli[8] found that injections of lecithin in cases of tabes and cerebrospinal lues changed a positive to a negative reaction in a large per cent. of the cases examined by him. In a few cases, however, the reaction increased in intensity.

Wolfsohn[9] found that after etherization a transitory positive reaction might occur in non-luetics. Narcotics, as alcohol and morphine, gave with us some positive results, both to cobra and Wassermann tests; but in no case could we exclude syphilis, or feel that the reaction was in any way due to the lipoid destroying power of the narcotic.

In a small number of cases, tabes, cerebrospinal lues and depression, a moderate, but not uniform, resistance to the hemolytic action of mercuric chloride was observed. In untreated primary cases this resistance was absent, but was present to a slight degree in untreated secondaries, although not as strongly as in those cases just stopping a three to five months' treatment. This change in cell resistance may be due to the generalized increased resistance to hemolysins produced as a reaction to the virus of lues, or it may be a possible adaptation of the cells to mercurial treatment. In experimental trypanosomiasis, a biologically related disease, resistance of the cells to drugs has been repeatedly observed. Experimental injections of hemolysins give a picture similar to that caused by the syphilis toxin; first anemia of a more or less severe grade, early fragility of the cells to hemolysins, followed by an increased resistance.

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[7] Weil, R., Jour. Infect. Dis., 1909, V, 688.

[8] Quarelli, Gaz. degli Ospedali e delle Cliniche, Milan, January 24, XXX, 1907.

[9] Wolfsohn, Deutsch. med. Wochnschr., March 17, 1910.

These changes probably affect the red cells while in the bone marrow, and not in the circulating blood; if so, other cells should be affected also by the virus. This may be one reason why mercurial treatment is ineffectual in parasyphilitic diseases.

To anisotonic salt, however, no clear cut resistance was found; if present it was accidental. Cells markedly resistant to cobra or other hemolysins, would often dissolve quickly in very slight deviations from the isotonic solution, and other cells showing little resistance to hemolysins would withstand marked dilutions of salt. The resistance to a specific circulating poison has probably a certain degree of specificity, and does not run parallel to resistance to anisotonic solutions.

The Much[10] reaction is the inhibition of cobra venom hemolysis of normal red cells, by the serum of manic depressive disease and dementia præcox. Using Much's technique, we obtained positive results in one case of cerebrospinal lues, two cases of general paralysis, one tertiary lesion of skin in a man with no mental disease in his history, one of exophthalmic goitre, one of confusional insanity, and in two cases of epilepsy. In only three out of thirty-seven cases of manic depressive disease did we get a positive response, but obtained positives in 62 per cent. of the hebephrenic type of dementia præcox, and 80 per cent. of the catatonic form. In studying the resistance of cells to hemolysins we found some differences in the behavior of cells of normal individuals to the dissolving action of cobra venom, even when cells had been washed the same number of times, at same speed, in same centrifuge, and revolved for the same number of minutes. We therefore modified the Much reaction by using for our indicator a cell whose resistance was such that it was entirely dissolved in a 1/5,000 solution of cobra venom in thirty minutes, at 37° C. With this modification we obtained positive results only in dementia præcox, 87 per cent., most strongly marked in catatonics, 100 per cent. All serums prevent in a slight degree the hemolytic action of cobra venom on corpuscles, but it is present in a much more pronounced manner in dementia præcox.

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[10] Much, Münch. med. Wochnschr., LVI, 1001, 1909.

The test is of some diagnostic value in dementia præcox, but only if *exactly* the same technique is used on every occasion with careful titrations of the venom and standardizations of the cell indicator. We could demonstrate no relation between the test and the Wassermann reaction, nor did it explain any of those cases of definitely non-luetic psychosis which gave weak positive or doubtful Wassermann reaction. Noguchi first called attention to these cases, and also to the fact that they gave positive results only with the serum, the reaction with the spinal fluid and butyric acid test being negative. We have so far never found a positive Wassermann and a positive butyric acid test, except when syphilis was present. In our non-luetic positive cases we repeated the test, using the plan proposed by Browning and McKenzie;[11] that is, to count as positive only those cases in which five more doses of complement are absorbed by the antigen-antibody compound than that absorbed by antigen and serum alone. Therefore, we first made a preliminary titration of the serum and antigen to determine the amount of complement absorbed by them alone. Many of the depressed mental states, which had given positive or doubtful reactions to the Wassermann technique, became negative by this empirical measure. The complement bound by the serum of all of our depressed cases varied between three and seven units, in one instance nine, and in no other condition so far examined by us have we found this high binding quality, although lately it has been reported in leprosy. It probably depends on the presence of some colloidal substance in the serum, and shows that chemical differences in the constitution of the blood sera may occur in mental disease. Normal cases never absorbed more than two units. Manic states were usually a little higher, between two and three. This titration may be of some diagnostic value. At present we state it very guardedly. The cases studied in the detention wards of the Philadelphia General Hospital and placed in the depressed group by the blood picture, later bore out the diagnosis psychologically. In all, sixty-seven cases of depression, irrespective of mental form of disease, have been studied, and all gave a high complement binding property.

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[11] Browning and McKenzie, Jour. Path. and Bact., 1908–9, XIII, 325.

In primary syphilis we found the most marked variations in results with the different syphilitic tests. An average of all of the first Wassermann examinations of our primary cases gave 60 per cent. positive results. The average of the fourth examination gave positive results in 98.5 per cent. This difference is probably due to several factors; the individual reaction to the virus, the virulence of the infecting agent, interference from the presence of complementoids, and the time elapsing between the infection and the examination. In most negative cases, subsequent examination at frequent intervals will sooner or later become positive. As in all biological reactions there are some cases that do not react; this is rare, but it detracts slightly from the value of the reaction. Illustration of this fact was the case of J. E. P. This man had an undoubted syphilis. He was examined first during the secondary eruption with negative findings. The disease was refractory, but finally yielded to very large doses of mercury. Three examinations were made during this latent period by different competent observers, and were in every case negative. His poor health at this time was thought to be due to over-mercurialization. One month after the last examination he developed convulsions and paraplegia. These cases, fortunately, are rare, but they emphasize the necessity of never estimating the value of any diagnostic laboratory test, except when taken in connection with all the other factors of the case.

In primary syphilis the presence of the spirochæte is the earliest positive sign. After it, a gradual steady increase in the globulins of the blood serum appears, and, at the same time, an increased fragility of the red cells to cobra venom and some other hemolysins. The Justus test for syphilis and the so-called mercurial test, we found corresponded to this period, and ceased to be positive when the cell resistance was firmly established. At the fourth or fifth week after the appearance of the chancre, the Wassermann reaction becomes positive, and increases in intensity as the disease approaches the roseola, or is modified by treatment. The fixation phenomenon is an indication that the infection has become generalized.

In the secondary stage the complement fixation test and the globulin increase are present in 98 per cent. to 100 per cent. of cases. Negative reactions, when they do occur, are generally the result of treatment, or very rarely to a refractory organism or lack of response in individual to stimulus of the virus. The fact is so rare that, unless very definite signs of syphilis are present, repeated negative findings should speak strongly against syphilis. The washed red cells acquire a marked resistance to cobra venom and some other hemolysins. The anemia is fully developed, the lowest hemoglobin count being reached coincidentally with the appearance of the rash. The hemoglobin remains low for a short time, and gradually rises as the exanthem begins to retrogress.

After the disappearance of the secondary, the patient enters upon a period of latency. Because of the frequent relapses of this disease much work has been done as to its cause. Virchow[12] thought the lymph glands formed the chief repositories for syphilitic virus from which the intermittent infection of the organism took place. Often, however, the relapse occurs where there is no demonstrable swelling of the glands. A mass of evidence based on repeated histological examinations has established the idea that, not only the glands, but every original seat of a lesion, may be a repository. While syphilis is a chronic dyscrasia, this by no means involves the necessity that the blood should at all times contain disease products. The latent periods of this disease may mean only that the organisms in the body, or floating in the blood stream, are so reduced qualitatively or quantitatively that they no longer are capable of evoking a lesion, or stimulating the production of antibodies, and this is entirely in line with the findings with the various tests. If, however, the resistance of the individual is lowered by trauma, infection, or other causes, this equilibrium between host and weakened spirochæte is lost, and the disease becomes manifest again. This manifestation may be simply a recurrence of the Wassermann reaction, or it may be tertiary lesion of the skin, bone, viscera, or nervous system. These localized residual foci of infection account for the non-symmetrical character of tertiary lesions, and for the smaller number of positive Wassermann reactions.

It is in these latent cases that we find the greatest value of the syphilitic tests. Often only one test is present, generally the globulin increase. In cases where there is a definite syphilitic history, a positive globulin result should not be disregarded. In three of our cases it was disregarded, and we were confronted in a short time with strongly positive Wassermann reaction, and in one case with a typical syphilide. In a fair proportion of the neurasthenic states, headaches, paresthesia, etc., a positive Wassermann reaction is obtained, and, very often, is the only definite sign of the disease. Treatment of the disease instituted at this period may save a patient from the more serious ravages of an active lues. Illustration of this is the case of F. M., who presented himself complaining of fatigue, depression and general nervousness. He had no demonstrable signs of syphilis, but a strongly marked positive Wassermann reaction. No syphilitic treatment was given and in a few weeks he appeared in the skin dispensary with typical tertiary lesion on hands and arms.

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[12] Virchow, quoted by Lesser, Beiträge zur klinischen Med., Festschrift Senator gewidmet.

The latent periods of syphilis become of very great importance also in the study of hereditary lues. A latent syphilitic with no clinical manifestations can give birth to a definitely syphilitic child. In the marriage of syphilitics the frequent study of the reaction is of utmost importance. Bar and Duanay[13] found that the percentage of positive results was higher in the off-spring of latent syphilitic mothers than in cases of florid syphilis, due to the fact that the latter cases were treated. We know that the Wassermann reaction disappears under treatment, and even under short courses, and that in a considerable number of cases it returns with more or less intensity after the cessation of treatment. Cases of general paralysis may arise ten or more years after an infection, late relapse may occur anywhere between five, ten, or twenty years after the initial sore, and the reaction has been found ten and thirty-five years after an infection. It becomes, therefore, difficult to say that a negative reaction, even if continued, means cure. The test is only in its fourth year, and sufficient time certainly has not yet elapsed to speak on this subject with any authority. It is still well to abide by the old laws for the marriage of luetics. Where pregnancy occurs in these marriages, the mother should be examined as early as possible in the pregnancy and thoroughly treated after the first positive findings.

Congenital lues gives a positive response to practically all the luetic tests. Under treatment the reaction weakens as the case improves, but not in a parallel manner. In only two of our cases it disappeared under treatment. The effects of congenital syphilis are many and far reaching. A moderate percentage of cases of convulsions, tremors, hydrocephalus, monospasms, paralyses, pseudo-paralyses, neuralgia, infantilism, imbecility, idiocy and progressive arrests of development, etc., give positive reactions. Cases of mental enfeeblement, especially where it ensues after a certain degree of intellectual development has been attained, are very often specific. In the majority of our cases of cerebral syphilis in children, we found some other evidences of lues, but a few had an entire absence of any stigmata.

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[13] Bar and Duanay, Obstetrique, Paris, March, 1909.

Mott,[14] in his third Morrison Lecture, quotes Fournier's classification of parasyphilitic affections due to hereditary syphilis. He tries to prove syphilis the fount and origin of almost every hereditary defect. Following his classification, we examined nine cases of dystrophies, seven of which gave negative results. Two other cases resulted positively; both of these children beside the dystrophy were distinctly feeble-minded, and the mothers gave history of miscarriages. In epilepsies, forty-nine cases, we found no case with a hereditary syphilitic basis, and obtained positive results only in those patients who gave a definite history and in whom the epilepsy followed the wake of the infection.

Among mentally defective or backward children, forty-nine cases, we obtained 36 per cent. positive results. Shuttleworth,[15] examining imbeciles for physical signs of syphilis, found only 1.17 per cent. positive. In the majority of our cases the children examined were not manifestly syphilitic. In some the mental backwardness and the Wassermann reaction were the only signs present. The tests help to the early recognition of the underlying cause and the removal of the toxic substance by proper treatment permits these children to develop.

Also Ziehen and Fournier[16] have years ago emphasized the improvement of defectives under mercury, and Joseph[17] cites cases which illustrate the great benefit which may follow treatment based solely on a positive Wassermann reaction, in dubious cases of inherited syphilis, normal except for some slight deviation.

In tertiary syphilis, whether it attacks skin, viscera or nervous system, the number of positive responses to specific tests falls very markedly from that observed in the secondary period, but they are much more frequent than in latent syphilis. This may be due to the weakening effect of treatment on the organism, so that it becomes unable to stimulate so markedly the antibody formation, or to a localization of the virus. In tertiaries the Wassermann reaction is of greater importance than in the earlier stages. In lesion of bones, five cases, the reaction has been with us strongly positive. Three were cases of multiple joint involvement, two of which gave also positive v. Pirquot reaction. In one case the father gave a positive history and positive syphilitic reaction. In another child an examination of the parents was not possible, but child improved in general health under mercurial treatment.

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[14] Mott, F. W., British Med. Jour., February 20, 1910.

[15] Shuttleworth, Am. Jour. Insanity, 1888, p. 381.

[16] Fournier, Syphilis Héréditaire Tardive, 1886.

[17] Joseph, Archiv. Kinderheil Kunde, April 17, 1909.

Winfield[18] reported a series of cases of bone disease in children, suggesting the possibility of hereditary syphilis as a predisposing cause for the development of the tuberculous process.

Swift[19] also reported the case of a man with a sinus of the rib and some softening of apices of lung. He was examined for tuberculosis and pathologist reported tuberculous tissue and tubercle bacilli. He gave a positive Wassermann reaction and the sinus healed under mixed treatment. It is well in cases of bone disease to submit the patient to both examinations; both infections occur together, the one probably making a very suitable soil for the growth of the other, or it may be possible that the tuberculous process calls to activity a latent syphilis by the stirring up of residual foci.

When tertiary syphilis attacks the central nervous system, the cobra venom test ceases to be of any material help, 22 per cent., the complement fixations appear only in about 67 per cent., and the globulin increase becomes the most constant sign, 94 per cent. The failure of the Wassermann reaction in these cases is a serious handicap, because it is often necessary to have some help in separating brain tumor or multiple sclerosis from cerebrospinal lues. Not infrequently also a cerebral tumor may arise in a syphilitic case and the positive reaction gives evidence of syphilis somewhere else in the body. In the majority of the cases seen by us, the individual had already had several courses of treatment, or was then under treatment with mercury and iodide; those seen in the beginning of their trouble gave much higher results. In eleven cases of genuine multiple sclerosis we obtained no positive results. In syphilis of the nervous system the value of the reaction has been very often described and its position definitely established. The reaction in these cases changes less easily under treatment, but very definitely. The strength of the reaction has no relationship to the extent of the disease.

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[18] Winfield, Jour. A. M. A., 1909, p. 1236, abs.

[19] Swift, Cleveland Med. Journal, May, 1910, p. 331.

In the parasyphilitic states again the globulin increase is the most constant finding; in these conditions parallel examinations of blood and cerebrospinal fluid are to be commended as one means of differentiating between tabes and taboparalysis.

The Wassermann reaction of the spinal fluid is a measure of the process of disintegration of the nervous system, or of grave disturbances in its metabolism. In tabes the complement fixation test is present in blood of 62 per cent. of cases. In cerebrospinal fluid in 40 per cent. of the cases, globulin increase in 98 per cent. In general paralysis, on the contrary, blood averages about 98 per cent. to 100 per cent., cerebrospinal fluid 98 per cent. to 100 per cent., globulin 98 per cent. to 100 per cent. If the difference in the blood and spinal fluid reaction, together with physical signs lately described by Fischer,[20] can separate a simple tabes from a tabes which will go into paresis, we certainly have a powerful factor for the peace of mind of many patients.

In the preparalytic stage of general paralysis it is of tremendous value for the protection of a family. During this stage a man's condition is masked by a neurasthenic syndrome. If there is any time in the history of paresis that specific medication may be of avail, it must be at this stage, when some intercurrent condition is first calling from abeyance to renewed activity those metabolic processes which were originally set up as a result of syphilis. Marie and Levaditi found a parallelism between the strength of the Wassermann reaction and the progress of the paresis. There was a correlation between the breaking down of nervous tissue and the amount of the colloid with which the reaction is associated, and on which it depends.

In mental conditions the Wassermann reaction has so far shown lues to have little etiological importance except in the cerebral manifestation of hereditary syphilis, general paralysis and syphilitic insanity. As an accidental complication it may, of course, occur in any psychosis. The Much reaction gave a high percentage of positive results in dementia præcox. The depressed conditions all gave an increase in complement absorbing power of serum and never gave a Much reaction. The colloidal substance present in these depressed cases does not influence the Much reaction, nor does it cause a true fixation of complement.

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[20] Fischer, Jour. A. M. A., August 27, 1910.

In conclusion we think that: The Wassermann reaction is enhanced in value by the parallel use of globulin tests and the cobra venom test; that: The Much reaction, by careful standardization, may be made of much diagnostic value in cases of dementia præcox.

Regarding the complement absorption of the serum, enough work has not yet been done to do more than guardedly report results. It is of interest in neurology, but final conclusion must be deferred.

THROMBOSIS OF THE CERVICAL ANTERIOR MEDIAN SPINAL ARTERY; SYPHILITIC ACUTE ANTERIOR POLIOMYELITIS[1]

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Thrombosis of the vessels of the spinal cord is a diagnosis that should be made more frequently, for while non-traumatic hemorrhage is rare, thrombosis is of frequent occurrence. With the exception of the case reported in this paper, I know of no instance in which the thrombosis was demonstrated by necropsy to be confined to a small portion of the anterior cervical supply. The case is also a contribution to syphilis as a cause of acute anterior poliomyelitis; to the position of the sensory tracts in the spinal cord; and to the pathology of brachial paraplegia. It demonstrates furthermore that occlusion of a limited portion of the anterior arterial system of the spinal cord or medulla oblongata produces a symptom-complex that should be capable of diagnosis even without necropsy, although the symptoms will vary with the level of the occlusion. The paper is in this way a further contribution to the subject of occlusion of the uppermost portion of the anterior spinal and adjoining portion of the vertebral arteries described by the author.[2]

John W., No. 456 Laboratory, had been employed in the Philadelphia General Hospital. He had been admitted in 1901 with symptoms of meningitis, but had recovered. On the morning of December 23, 1905, he was lifting blocks of ice with the assistance of another man, each block weighing about 100 pounds. He had lifted four pieces when about eight o'clock A. M. he began to have a sensation of coldness and pain between the shoulders about fifteen minutes after lifting the last piece. The hands began to grow stiff and he placed them under the spigot, allowing hot water to run over them. Ten or fifteen minutes later the lower limbs began to be numb and stiff. He lost power in the upper limbs within a very short time.

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[1] Read at the Thirty-fifth Annual Meeting of the American Neurological Association, May 27, 28 and 29, 1909.

[2] Spiller, The Journal of Nervous and Mental Disease, December 1908.

Notes made on December 26 state that he was totally paralyzed in the right upper limb and had not a single movement of hand, forearm, fingers or upper arm. The left forearm could be flexed on the arm, but could not be extended, and he had some power of elevation of the shoulder. The lower extremities were weak in all movements, but there was no pronounced palsy.

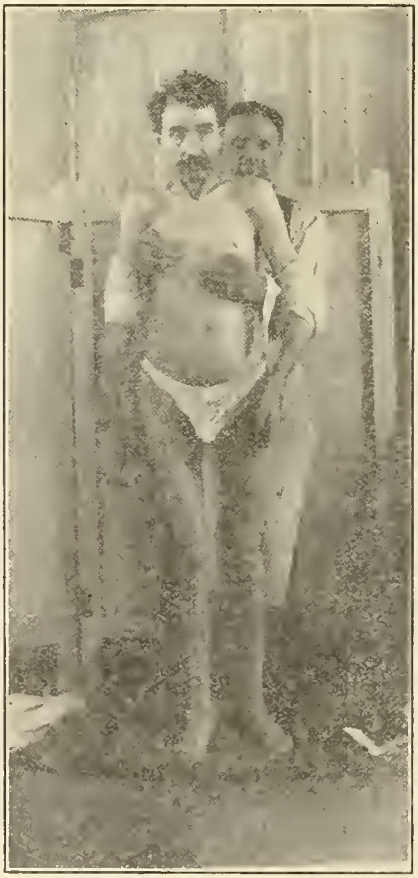

Fig. 1. The man is not held up, the orderly's hands are barely touching him. He is bearing full weight on his lower limbs.

Breathing was entirely diaphragmatic. Hypalgesia was noted between the nipples and clavicles and in the upper extremities. He had retention of urine with overflow, and no control of the bowels. The patellar reflexes were preserved and the Babinski sign was not obtained.

Notes made by me January 22, 1906, are as follows: The voluntary power in the lower limbs is not much diminished. The patellar reflex is prompt on each side. The lower limbs are emaciated to some extent. Babinski's reflex is slight on the right side, but there is no movement of the big toe on the left side. Sensation for touch is normal in the lower limbs. Pain and temperature sensations are much affected in front of each thigh, especially in the left, and to some extent on the back of the left thigh. Sensation below the knees is unaffected, except on the sole of the left foot, where sensations of pain and temperature are impaired.

Sensation for touch is normal in the trunk and upper limbs. Sensations of pain and temperature are greatly impaired, if not lost, over the whole of the front of the trunk, as high as the third rib on each side, also in both forearms as far as the elbows, and on the inner side of the left arm above the elbow, although the disturbance is not so great as in the trunk. He has incontinence of urine and feces.

The notes of an examination by Dr. Knipe January 26, 1906, are as follows: O.D. pupil 5 mm. Iris reacts freely to light. O.S. pupil 4 mm. Iris reacts rather sluggishly to light. O.S. reacts more freely to convergence. O.S. pupil smaller on account of iris infection (?), iris is more cloudy than iris of O.D. O.D. media clear and fundus negative. O.S. media clear and fundus negative. Ocular movements unimpaired.

Further notes made by me March 19, 1906, are as follows: The lower limbs are emaciated, but equally in all parts, and the limbs are somewhat spastic. The patellar reflex is much exaggerated on each side and equally, although the right may be a trifle more so than the left. Patellar clonus is persistent on the left side, but can not be obtained on the right side except when the muscles are slightly relaxed, as it is prevented on this side because of the rigidity of the muscles. Ankle clonus is not obtained on either side. Achilles tendon reflex is not obtained on either side. The Babinski reflex is present and typical on the right side, but is uncertain on the left side. The movements are free in all parts of the lower limbs, and resistance to passive movements is good in these limbs. Involuntary jerkings of the lower limbs occur, especially of the right lower limb. He is able to stand and walk unaided, even with the eyes closed. The right lower limb is slightly weaker than the left.

The man knows when he wishes to pass urine, but he can not hold the urine long. Sense of position is preserved. Breathing is almost entirely diaphragmatic, and he hardly raises the ribs at all. He can not move the fingers of the right hand at all, and he has very little power of motion at the right shoulder and right elbow. He has a sore on each shoulder from friction. The right fingers are flexed into the palm, the right thumb is extended. The fingers can not be forcibly fully extended. The muscles of each forearm and of the upper part of the arms are flabby and wasted. There is tendency to flexor contracture at the right wrist which is readily overcome by passive movements. No contracture at the right elbow. The right triceps and biceps reflexes and wrist reflexes are entirely lost. No tenderness to pressure anywhere.

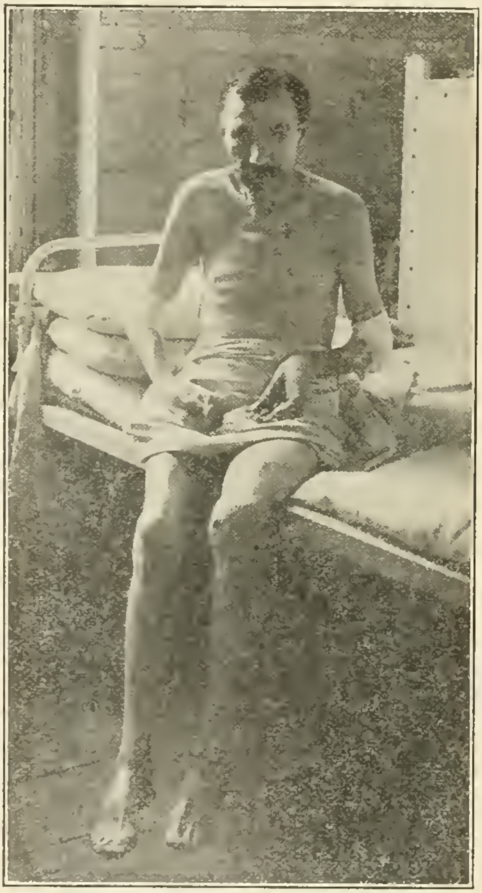

Fig. 2. The contractures of the upper limbs are well shown.

The fingers of the left hand are very slightly flexed and can be fully extended. The left hand is contractured in over-extension at the wrist, and is in contrast to the flexor contracture of the right hand. The contracture of the left hand can be easily overcome by passive movement, as the contracture in each hand is only in an early stage. Marked flexor contracture has occurred at the left elbow, this can be overcome with difficulty, and the attempt to do so causes considerable pain. He can raise the left upper limb only at the shoulder, and performs this movement chiefly by action of the trapezius, the deltoid functioning very little; on the right side the deltoid is a little more active than on the left.

The cranial nerves are normal. The right pupil is larger than the left. The reaction to light and in accommodation and convergence is prompt in each eye. The movements of the eyeballs are normal. The movements of the head are free in all directions.

Sensations of heat and cold are normal in the whole right lower limb, but are greatly impaired in the whole left lower limb; here both ice and warm water are felt as warm. These sensations are greatly impaired over both sides of the trunk as high as the first rib on the left side, and as high as the second rib on the right side, and in the entire left upper limb as high as the clavicle. They are considerably impaired in the right upper limb, and both heat and cold are felt as heat. Sensation of touch is normal in all parts of the body. Sensation of pin-prick is normal in the right lower limb, except in the uppermost part of the thigh near the trunk and middle of the thigh, where it is impaired. It is greatly impaired in the left thigh, and much less so in the left leg below the knee. It is greatly impaired in the trunk and left upper limb in the same distribution as is temperature sensation. It is much impaired in the right upper limb except near the shoulder. Sensations of touch, pain and temperature are normal in the face.

It was recorded on May 9, 1906, that the man was walking about the ward alone, and did so fairly well. He died November 1, 1908, in the service of Dr. Charles K. Mills, and to him I am indebted for the pathological material.

The first appearance of softening begins at the fourth cervical swelling. A cavity at the highest part of this segment is confined to one anterior horn, but at a slightly lower level both anterior horns show softening, with cavity formation at the posterior part of each horn, extending slightly into the white matter. The antero-lateral portion of each side of the section shows secondary degeneration, as does also the anterior part of the posterior columns, in which a small collection of fatty granular cells is found. Nerve cells are present in both anterior horns and are more numerous in the right anterior horn.

At the fifth cervical segment the cavity is again confined to one anterior horn, otherwise the condition is much the same as in the fourth cervical segment. Both anterior horns contain nerve cells, and they are more numerous in one than in the other. A blood vessel in the posterior septum near the commissure shows great thickening and some round cell infiltration.

At the sixth cervical segment each anterior horn contains a separate area of softening with many fatty granular cells, and the whole anterior part of the cord, including the anterior part of the posterior columns, presents secondary degeneration. The anterior horns at this level contain few nerve cells.

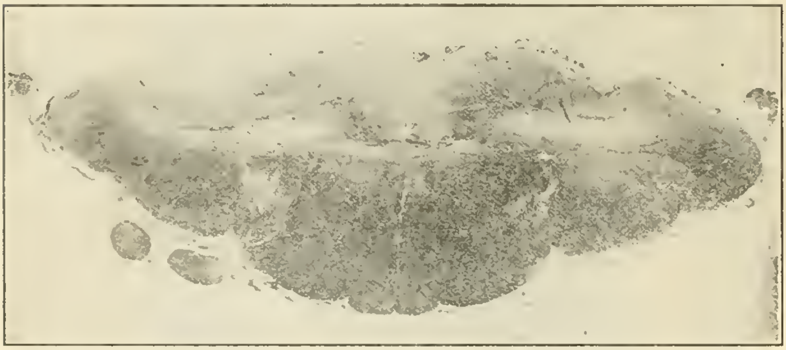

Fig. 3. Photograph of a section from the seventh cervical segment, showing intense degeneration of the anterior part of the cord. The pyramidal tracts are almost intact.

The condition is much the same at the seventh cervical segment, but at the eighth cervical segment the areas of softening are more extensive; on one side extending to the anterior fissure, and on both sides extending somewhat beyond the anterior horns into the white matter. The crossed pyramidal tracts are slightly affected, but only in their anterior portion.

At the first thoracic segment the softened area forms one lesion, occupying the entire anterior part of the cord in front of the anterior horns. This is the region of greatest destruction. Some of the vessels in the anterior part of the pia at this level show much thickening, and the anterior spinal artery has great proliferation of its intima. Some of the vessels in the area of softening at this level are almost completely occluded.

The area of softening becomes less extensive at the second thoracic segment, and disappears entirely at the upper part of the third thoracic segment.

Above and below the region of softening secondary degeneration is seen. The crossed pyramidal tracts are partially degenerated into the lumbar region.

Round cell infiltration is moderate in intensity in the cervical swelling, and slight elsewhere over the cord. It is more pronounced in the pia of the optic nerves and medulla oblongata.

The anterior roots of the cervical swelling are much degenerated.

The occlusion of the blood vessels of this case occurred only in the lower cervical and first thoracic segments, and only in these regions were the blood vessels distinctly thickened, and here only in marked degree in the anterior part of the cord. This proliferation of the intima with the wide-spread though moderate round cell infiltration of the pia is strongly indicative of syphilis.

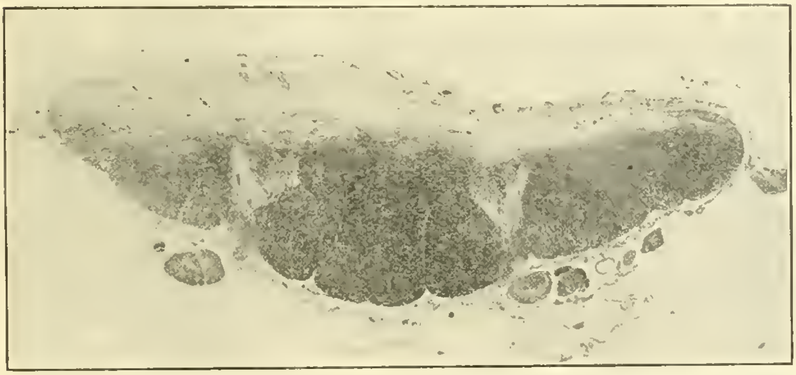

FIG. 4. Photograph of a section from the first thoracic segment, showing complete degeneration of the anterior part of the cord. The posterior part of the cord is almost intact.

A summary of this case is as follows: About four years previous to his paralysis the patient had what was supposed to be spinal meningitis. He recovered, but probably had as a result thickened blood vessels of the spinal cord. He had been in excellent health, according to his own statement, and was employed at the time the paralysis occurred in an ice house, and with another man had been lifting blocks of ice weighing about 100 pounds. He had lifted four of these blocks before any symptoms were noticed. Fifteen or twenty minutes after lifting the last block he began to have pain between the shoulders, and felt numb and weak in the upper extremities. Ten or fifteen minutes later he felt weak and numb in the lower limbs, and was then brought to the hospital. When first seen by me in January, 1906, he had recovered largely the use of the lower limbs, but these limbs were somewhat spastic. He had normal sensation of touch everywhere. The patellar reflexes were exaggerated and Babinski's reflex was present on the right side. He had much impairment of temperature and pain sensations in the upper part of the thighs, over the trunk as high as the first or second rib, and in both upper limbs. He had incontinence of the bladder and rectum. He was almost completely paralyzed in the upper limbs, and these limbs were much wasted. The case was reported before the Philadelphia Neurological Society as one of probable hematomyelia of the cervical enlargement. There seemed to be little doubt that the lesion must be in the cervical enlargement and first thoracic segment, as it would be impossible to explain the symptoms in any other way. It was believed to be a lesion affecting the gray matter of the anterior horns and extending into the white matter. The posterior portion of the cord, from the symptoms, must have escaped. The investigations of Minor have shown that central hematomyelia causes the dissociation of sensation as does syringomyelia, and the rapidity of development of the symptoms suggested hemorrhage. A probable thickening of blood vessels was mentioned, but as no case is on record, so far as I know, in which thrombosis was confined to the distribution of a small part of the anterior spinal arterial supply, with such symptoms as occurred in this case, one may be pardoned for diagnosing hemorrhage in place of thrombosis. Minor,[3] says that in the many cases of hematomyelia observed by him the hemorrhage occurred spontaneously only in two cases, in all the others trauma seemed to be the cause.

The microscopical examination has shown that the lesion was thrombosis. The anterior spinal artery and the branches coming from it, in the eighth cervical and first thoracic segments were much thickened, and some of these vessels were almost entirely occluded. They were doubtless entirely occluded in areas not obtained in the sections. The softening was intense in the first thoracic and eighth cervical segments, as shown by the presence of numerous fatty granular cells and a few minute hemorrhages. The anterior horns were softened above these regions as high as the fourth cervical segment. The lesion implicated the anterior horns, and the whole anterior part of the cord in advance of the crossed pyramidal tracts, and the extreme anterior part of the posterior columns. The pyramidal tracts were partially degenerated. As far as location is concerned the clinical diagnosis was exact. The round cell infiltration of the pia, moderate in the spinal pia and more pronounced in the cerebral pia, together with the proliferation of the intima of the anterior spinal artery and its branches in the lower cervical swelling, indicate syphilis as the cause of the lesions.

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[3] Minor. Handbuch der pathologischen Anatomie des Nervensystems, Vol. 2, p. 1,031.

The case is extremely important in showing that thrombosis may occur in a very limited distribution of spinal vessels. We hear little of spinal thrombosis, although we are very familiar with thrombosis of the cerebral vessels. Spinal thrombosis is not a rare condition, and is probably the cause of most of the apoplectiform palsies that occur in myelitis. Then the occlusion is usually widespread, or at least implicates most of the vessels in any one transverse region of the cord. In the case reported by Dr. Mills and myself,[4] in 1903, paralysis of all four limbs and of one side of the face developed in about five hours. Some of the blood vessels of the cervical swelling had much thickened walls and some of those in the pia at this level were almost occluded by the intense proliferation of the intima. Thrombosis was doubtless the cause in this case of the rapidly developing paralysis.

That such palsies occur frequently is well known and it seems unnecessary to devote much space to the demonstration of this fact, and yet two cases recently observed by me may be alluded to briefly in way of illustration: E. M., aged 27 years, had had slight ataxia occasionally in walking about six months before any weakness developed, but he had not had any loss of motor power. He sat down in a chair on one occasion, and after about fifteen minutes tried to rise but was unable to do so. He had to be assisted from the chair and was in bed a week before he could stand at all. He has now symptoms of postero-lateral sclerosis.

Mrs. C. C., aged 28 years, had deserted her husband because he had venereal disease. She had been walking much on a certain day, and about nine o'clock in the evening of this day started to go to bed and found that she was weak in the knees, and that she could not empty the bladder. At nine-thirty she walked to the bath-room, and on returning fell to the floor and was at once unable to stand even with support. She is now very weak and spastic in the lower limbs. I could add to these the reports of other cases of very rapid paralysis in myelitis, some with necropsy, but it seems unnecessary to do so.

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[4] Mills and Spiller. JOURNAL OF NERVOUS AND MENTAL DISEASE, 1903, p. 30.

I know, as previously stated, of no example recorded in which a small part of the anterior median spinal artery with its branches were so clearly shown to be the seat of thrombosis as in this case I now report. Thrombosis is possibly the cause of the symptoms in acute anterior poliomyelitis, as has been emphasized especially by Batten,[5] although he has not originated this view. He believes that this disease is caused by primary thrombosis of a branch or branches of the anterior spinal artery supplying the gray matter of the anterior horns, and that such thrombosis may be produced by many and various forms of infection, and that the disease is not due to a specific infection. The condition is more likely to occur in the lumbar region owing to the blood supply of this portion of the cord being at a point most distal from the heart, and to the long course of the reinforcing arteries.

He refers to the work of Prevost and Cotard, by which it was shown that congestion, hemorrhages and exudation of cells may be the early result of obliteration of small vessels. Batten refers to the well-recognized fact that the lesion conforms closely to the distribution of the anterior median artery, and he thinks it is reasonable to conclude that it is the result of occlusion of vessels. This view, however, is not generally accepted.

More recently syphilis as a cause of acute anterior poliomyelitis has been discussed. Preobraschenski[6] states that the anterior horns are often affected in spinal syphilis, and the condition has been described as chronic or subacute poliomyelitis, as parasyphilitic muscular atrophy, or as syphilitic amyotrophy of the Aran-Duchenne type. He states also that all the cases described under these names presented much that was atypical both clinically and anatomically for progressive muscular atrophy or poliomyelitis, but characteristic of syphilis. He refers to

[5] Batten. Brain, 1904, p. 376.

[6] Preobraschenski. Neurologisches Centralblatt, November 16, 1908, p. 1,069.

cases reported by Dejerine, Schmaus, Dinkler, Raymond, Leri, Olivier and Halipre, Lannois and Parot. He states that paralysis with later atrophy is common from syphilitic meningomyelitis and hardly worth discussion. In all the cases referred to muscular atrophy and paralysis were more or less permanent, but were not the only signs. In almost all the cases the findings were diffuse alteration of the cerebral membranes, of the vessels, of the white and gray substance, of the roots, etc.

The case that Preobraschenski reports is as follows: A woman, 46 years old, who probably had acquired syphilis and had used much alcohol, felt on one night great weakness of all the limbs; the weakness increased. The exact period of the development of the weakness is not stated clearly. The cranial nerves were normal. Pupils were equal and did not react to light. Both upper limbs and the right lower limb were completely paralyzed, and the left lower limb was almost paralyzed. The head alone could be moved freely. All the muscles were flaccid. The tendon reflexes were normal in the left upper limb, were weak in the right upper limb, and lost in both lower limbs. Babinski's sign was not obtained. Sensation was normal. Pressure on the nerve trunks of the upper and lower limbs was painful. The urine was not passed for a time. The paralyzed muscles became atrophied. Death occurred on October 4, '07, the patient having been admitted to the hospital on July 26, '07, two days after the beginning of the symptoms. The case seemed clinically to be one of polyneuritis with paralysis of the abdominal muscles without sensory disturbances. The microscopical examination showed acute poliomyelitis, extending from the cervical swelling to the end of the cord. The thickening of the vessels, the round cell infiltration, the implication of the pia, indicated syphilis. Preobraschenski regards his case clinically and pathologically as a pure example of acute poliomyelitis, and states that the text-books of neurology and the literature in general make no mention of syphilitic acute poliomyelitis; his case in his opinion fills the gap. He acknowledges that one case is not sufficient to establish a symptom-complex, and that further investigations are necessary. Symptoms of cerebral origin are to be expected.

The case that I have reported in this paper differed clinically from acute anterior poliomyelitis in the sensory symptoms, as these are absent in poliomyelitis, and yet the lesion was acute, was thrombosis of the anterior spinal arterial supply, and caused the sensory symptoms by extension of the lesion into the white matter of the antero-lateral columns. It would seem either that in some cases the distribution of the anterior spinal artery may be more extensive than in others, or that the escape of the white matter so far as indicated by the absence of sensory symptoms is caused by less involvement of the branches extending into the white matter. We must believe that the branches of the anterior spinal artery extend at least a certain distance into the white matter. This case of mine was not therefore a typical one of acute anterior poliomyelitis clinically, and yet it affords considerable support for the views of Preobraschenski.

Observations made during the recent epidemics of poliomyelitis, that have been so numerous, have shown that our views regarding this disease needed broadening. In some cases pain has been so prominent that neuritis has been seriously considered, and to many the association of multiple neuritis with poliomyelitis seems probable. I[7] have published the report of two cases of poliomyelitis, with exaggeration of the patellar reflexes, and have referred to a few similar cases (Pearce, Clark and Atwood, Minor). In another of my cases of poliomyelitis a necropsy was obtained and considerable involvement of the lateral columns was found, although the degeneration of the anterior horns of the mid-lumbar region prevented exaggeration of the patellar reflexes. Had the lesion of the anterior horns been confined to the sacral region we should expect exaggeration of these reflexes in such a combination of lesions, and yet loss of tendon reflexes has been regarded as one of the most striking features of anterior poliomyelitis.

The lesions in this case afford evidence that the fibers of pain and temperature ascend in the antero-lateral columns, possibly but not necessarily in the tracts of Gowers, and that the fibers of touch ascend in the posterior columns. This subject has been fully discussed by Petrén,[8] and I[9] have written on it elsewhere.

Investigations have shown that Gowers's tract does not terminate entirely in the cerebellum and that a part enters the cerebrum. The whole anterior portion of the lower cervical sections was degenerated, pain and temperature sensations were greatly affected in the trunk and upper limbs, while tactile sensation was normal everywhere. The partial escape of pain and temperature sensations in the legs below the knees may be explained by the "law of the eccentric and posterior position of the long fibers," as these fibers in the lateral columns are probably the most posterior of those conveying pain and temperature sensations. As the posterior columns were intact, except in their extreme anterior portion, the escape of tactile fibers is readily explained, *i. e.*, as the result of the integrity of these columns. This case seems to be another proof of the representation of tactile fibers in the posterior columns. Petren believes however that tactile fibers are represented also in the lateral columns.

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[7] Spiller. JOURNAL OF NERVOUS AND MENTAL DISEASE, 1908, p. 261.

[8] Petrén. Skandinav. Archiv f. Physiologie, Vol. 13, 1902.

[9] Spiller. University of Penna. Medical Bulletin, July and August, 1905.

I have observed a number of cases of persistent brachial paraplegia with at first impairment of the motor power of the lower limbs, and later complete or nearly complete recovery of power in these limbs, but necropsy in this type is exceedingly rare, and the case J. W. is therefore an important contribution to the subject.

I am indebted to Dr. A. J. Smith for the photographs of the spinal cord.

EPILEPSIA PARTIALIS CONTINUA OCCURRING IN CEREBRAL SYPHILIS

REPORT OF A CASE, WITH OPERATION *

WILLIAM G. SPILLER, M.D.
Professor of Neuropathology and Associate Professor of Neurology in the University of Pennsylvania

WITH THE COLLABORATION OF

EDWARD MARTIN, M.D.
Professor of Clinical Surgery in the University of Pennsylvania
PHILADELPHIA

It seems almost incredible that any form of epilepsy should exist and yet not be well recognized. The literature of this disease is very extensive, and much of it contains little that is new. There is a type of epilepsy, however, which does not appear in the text-books, and even in the most recent writings receives scant recognition. Thus, Oppenheim[1] mentions it in a few lines by stating that cases occur in which during the interparoxysmal period muscular twitchings of short duration and without loss of consciousness are observed in one or another portion of the body. It has been described, especially by Russian writers, as "epilepsia continua" (Koshewnikow, Muratoff, Bechterew); Bruns also mentions it.

The cases differ from those of myoclonic epilepsy; the twitchings are limited to a small portion of the body, and are more intense than in myoclonia.

The name, "epilepsia partialis continua," seems to have been first employed by Koshewnikow,[2] who reported four cases in 1894. Characteristic epileptic attacks occurred in all four cases; sometimes they were abortive, sometimes they were with loss of consciousness. No period of cessation occurred between these at-

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* From the Philadelphia General Hospital and from the Laboratory of Neuropathology of the University of Pennsylvania.

[1] Oppenheim: Lehrbuch der Nervenkrankheiten, ed. 5, p. 1389.

[2] Koshewnikow: Neurol. Centralbl., 1895, p. 47.

tacks, as in certain portions of the body clonic movements persisted with variation in intensity, and when these movements became very severe they passed into an ordinary epileptic convulsion. The patients were unable to work, as they held the twitching limbs with those not implicated by the movements. In two of the cases the disorder began with local spasm, which passed into the ordinary epileptic convulsion; in the other two the persistent clonic movement followed the ordinary epileptic attack. No information as regards pathology was obtained either by operation or necropsy. This form of epilepsy has, since the report of these cases, been called, by some writers, Koshewnikow's epilepsy.

Orlowski[3] reported a case of this type in which general epileptic attacks occurred after an infectious disease, and later the patient had attacks every ten or fifteen minutes without loss of consciousness. Twitching of fibrillary or tic-like character was seen in the right side of the face, implicating also the tongue and larynx and continuing during sleep. The right limbs were weak. The case was thought by some of those who heard the report to be functional and not organic. No pathologic cause was determined.

Choroschko has written a monograph on this form of epilepsy, but, as it is in the Russian language, I have been obliged to depend on abstracts.[4] In the *Jahresbericht für Neurologie und Psychiatrie*, 1907, it is stated that Choroschko reports three cases of his own and refers to eleven in the Russian literature. Outside of Russia, Bruns alone has observed this rare type. Choroschko differs from Koshewnikow in that he believes the lesion is subcortical and suggests the name "polyclonia epileptoides continua." The twitchings are clonic and extend gradually from a limited area, and usually synergetic muscles are affected, especially on the right side. There seem to have been no lesions found either by operation or necropsy, so far as one may judge from the abstracts.

It may not be amiss, therefore, to report a typical case of this partial constant epilepsy, especially as it is one in which operation was performed. It shows that the lesion may be cortical.

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[3] Orlowski: Neurol. Centralbl., 1896, p. 526.

[4] Choroschko: Neurol. Centralbl., 1908, p. 279; Jahresb. f. Neurol. u Psychiat., 1907, p. 772.

History.—The patient, a colored man, aged 22, was admitted to my service at the Philadelphia General Hospital, Feb. 19. 1908. He gave a clear history of syphilitic infection. In July, 1908, he had his first convulsion. The fingers of the right hand twitched at first slightly but later more severely, and the convulsion confined to these members lasted about twenty minutes and left a paralysis of the hand persisting about fifteen minutes. He had another convulsive attack confined to the right hand in about three weeks. The whole right upper limb became implicated in the third attack, and this attack was followed by unconsciousness. There seems to have been involvement of the face and right lower limb also in the fourth attack. After these repeated attacks persistent weakness of the right upper limb ensued.

Examination.—The weakness of the right side of the face increased greatly after the man entered the hospital, and the palsy was of the cerebral type, i. e., slight in the upper part of the face and almost complete in the lower part. The weakness of the right upper limb also increased considerably, and the right biceps and triceps reflexes were much exaggerated, whereas the corresponding reflexes on the left side were normal. Stereognostic perception and sense of position were lost in the right hand. Tactile sensation also seemed to be diminished in this part, but was normal in the face. The motor power was about normal in the lower limbs, and the patellar reflex was only a little prompter on the right side. Babinski's sign was not distinct on either side The ocular examination revealed little that was abnormal.

The case was clearly one of syphilis of the brain with convulsions of the Jacksonian type, only the right side of the body being involved, and the persistent paralysis being confined to the right side of the face and the right upper limb. Other attacks which the patient had made the case an unusual one and worthy of careful study.

Independently of the typical attacks he had twitching confined to the right side of the face lasting an indefinite period, an hour or more at a time and occurring very frequently. There were periods in which he had none of these spasmodic movements, but the movements were almost constantly present, and resembled a tic movement or fibrillary twitching. They were not excessive in their intensity, and implicated the muscles about the right side of the mouth, about the right eye, the right cheek, the right ear, and the right side of the neck. After persisting for some weeks in the face they ceased in this part, and were observed in the right upper limb. The whole right upper limb was displaced in these clonic movements, but the displacement was not excessive, and the spasm consisted of slight twitchings lasting indefinitely as did the previous twitchings of the right side of the face. There was no impairment of consciousness. The peculiarity was the almost constant duration of fibrillary or twitching movements in the face at first and later in the right upper limb, movements that were very unlike the severe spasmodic contractions usually seen in an epileptic attack, both in intensity and duration.

It is a question whether operation is advisable in cerebral syphilis. Two cases with operation have been observed by me in addition to the one described in this paper; in one the removal of a gumma of the dura was followed by much improvement during a period of seven or eight years, but the patient eventually died of his syphilis. In another case excision of a gumma of the brain was performed, but the result was far from satisfactory, and in the case reported in this paper operation accomplished little. However, as there seemed to be a chance for the patient from operation, I referred him to Dr. Edward Martin, whose report is here given:

Because of the failure of mercury and iodid treatment, the steady progression of symptoms, the possibility of their leading to a fatal termination, operation was deemed advisable in this case. The Rolandic area was exposed by a periosteal flap which was raised by means of a Crile cutting tool driven by an electric engine. The dura was raised without difficulty, exhibiting only slight adhesion to gummata placed in the arm and face centers. A portion of this tissue was excised by circular excision, the obvious vessels leading to it being first encircled by fine gut carried by means of a fine pointed curved needle. The operation was attended by trifling shock.

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DISTRIBUTION OF ENCEPHALIC HEMORRHAGES[1]

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INSTRUCTOR IN NEUROLOGY AND NEUROPATHOLOGY, AND LECTURER ON ABNORMAL PSYCHOLOGY, UNIVERSITY OF PENNSYLVANIA, PHILADELPHIA

With Dr. Beevor's drawings of the areas of the brain supplied by the different arteries and with injected specimens of my own, comparison has been made with the points of softening and areas involved by hemorrhage in the cases collected in the laboratory of Dr. Spiller.

Accurately mapped out areas of the distribution of the various cerebral vessels have not been presented prior to the publications of Dr. Beevor[2] and that work has made it possible to definitely establish the location of vascular lesions. These lesions have usually been described as originating in the striate group of vessels, but it would seem in the 93 cases examined in this laboratory that they occur about as frequently in the anterior choroid, the posterior communicating, and the posterior cerebral arteries. They all are important factors both in softenings, cyst formations and hemorrhages in the region of the basal ganglia.

The blood supply of the internal capsule is from the anterior cerebral, the striate vessels, the posterior communicating, and the anterior choroid vessels.

The caudate nucleus has a supply from the anterior cerebral tion of fibrillary or twitching movements in the face at first and later in the right upper limb, movements that were very unlike the severe spasmodic contractions usually seen in an epileptic attack, both in intensity and duration.

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[1] From the Department of Neurology and the Laboratory of Neuropathology in the University of Pennsylvania. Read at the thirty-fifth annual meeting of the American Neurological Association, May 27, 28 and 29, 1909.

[2] Philosophical Transactions of the Royal Society of London, Series B, Vol. 200, 1908.

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The blood supply of the internal capsule is from the anterior cerebral, the striate vessels, the posterior communicating, and the anterior choroid vessels.

The caudate nucleus has a supply from the anterior cerebral and the striate vessels, as can be seen in the diagrams of Dr. Beevor.

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The thalamus does not receive a supply from the striate group, but does from the posterior cerebral and posterior communicating.

The lenticular nucleus segments receive small quantities from the anterior cerebral and anterior choroid, the greater part, however, being from the striate group of vessels. As is shown in the drawing (Part A) of the injection of the middle cerebral artery there is a sharply demarcated line between the circulation of the internal capsule and the lenticular nucleus. This line shows the internal boundary of the supply of the middle cerebral and agrees with the line drawn by Dr. Beevor.

This other injection shown in the diagram (Part B) is of the striate group of vessels of the middle cerebral artery, the remainder of the artery being tied off.

It shows that the internal border is the same as in the other injection but that it does not laterally go beyond the claustrum. These two injections alone are sufficient to demonstrate that there is no anastomosis in the terminal branches of the cerebral arteries, and that the area supplied by one artery is distinct from any other.

Areas of softening in our cases here collected usually occurred at these boundary lines, which is explained by occlusion of a terminal branch by sclerosis or other cause.

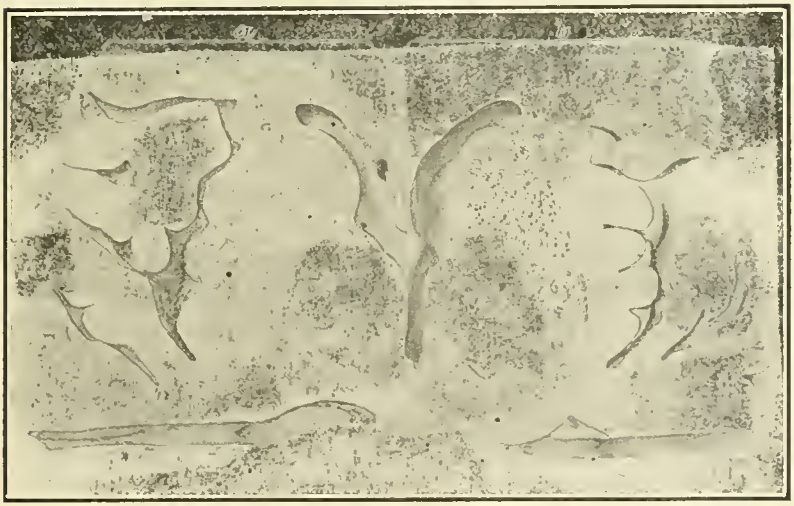
Softening has been found the most common condition; among 93 lesions studied, 69 were softened areas, and in many cases of hemorrhage it was possible to demonstrate the initial lesion as a softened area.

The posterior cerebral artery plays more of a part in these conditions than is commonly considered. I found 7 cases of hemorrhage and 6 cases of softening in the distribution of this vessel in the thalamus, and in most of the cases it was possible to demonstrate the point of rupture of hemorrhage. The basilar distribution in the pons was responsible for six lesions. There were eight in the posterior communicating arterial areas in the crus and thalamus. The anterior cerebral had two at the extremity of the caudate nucleus.

The anterior choroid artery had three lesions in the internal capsule in the region which is supplied by that artery, as shown by the charts of Dr. Beevor.

The remainder of the lesions involving the internal capsule were from the striate group of vessels, having erosions in the lenticular nucleus extending over into the internal capsule, while the majority of lesions are in the area of the striate vessels, and situated in the lenticular nucleus and external capsule; yet many heretofore considered as being from the striate group are not so in reality.

The thalamus, according to Beevor and substantiated by all the injections which I have made, does not receive the terminal branches of a so-called lenticular optic artery.



Part A. Injection of entire middle cerebral artery. Shaded portion is the injected area.

Part B. Injection of striate vessels with Higgin's red drawing ink, shaded portion. No anastamosis with surrounding parts.

All the branches going through the anterior perforated space ascend posteriorly to the anterior commissure; they quickly divide and supply the major part of the lenticular nucleus and the greater part of the caudate, they do not supply any tissue external to the external capsule, and the drawings show that these striate vessels do not anastomose at this line with the remainder of the middle cerebral vessels.

Two pathological specimens in our laboratory show this very distinctly, one is a thrombosis of the middle cerebral beyond the departure of the striate vessels, and with consequent atrophy and degeneration of tissue extending ventrally to the claustrum. The other specimen is the reverse picture, that is, a softening of the entire striate region. The external border of this area corresponds exactly with the internal border of the thrombotic area.

The striate vessels vary in size from all very small ones in some brains to a fewer number and larger in others, and in their path upward no one seems any larger than any other, and I feel sceptical about giving the name lenticular striate or lenticular optic to any one artery, and calling it the artery of cerebral hemorrhage, notwithstanding the great authority of Charcot.

In all the cases of hemorrhage there was no artery sufficiently larger than another to be worthy of a name peculiar to itself, and the hemorrhage and softenings were in different areas in each case, and when hemorrhages were found in the thalamus they could not be designated as coming from a so-called lenticulo-optic artery, but were really from the posterior cerebral vessel.

And too, the anatomy of the striate vessels is not at all constant; in two distinct cases a large branch of the anterior cerebral passed backward and ascended in the anterior perforated space and acted as a striate vessel.

The physical reasons given by Duret and Brissaud as the causative factors in hemorrhage are: first, vascular hypertension; second, defect in consistence of parenchyma; third, defect in resistance of artery. For those vessels which are described as being the ones of predilection the explanation is, that they are small, fragile arteries arising directly from large and strong vessels, without the usual graduated transition in size; also that they do not anastomose among themselves, or with other vessels, and that with a pressure equal to that in the carotids these fragile terminal vessels readily rupture.

Pressure in extensible tubes produces not alone dilatation but elongation, this fact taken into consideration when there is sclerosis of the hexagon of Willis and of the small vessels, makes it quite clear that repetition of the elongation and dilatation with every systole would in those vessels unprotected by graduated transition in size make them prone to early rupture.

This type is, however, not confined to the striate vessels alone, but the conditions are true of the branches of the posterior communicating going to portions of the thalamus and other parts, and of the anterior choroid going to a part of the internal capsule, optic tract, peduncles and other structures. This latter vessel is somewhat relieved by the anastomosis of the main branch with the choroid plexus.

The line of the external capsule is the area of predilection, so termed by Charcot; it is the line where the striate circulation meets the other branches of the middle cerebral, and it is quite similar to all other areas where lesions occur, in that, a shutting off of the blood supply causes an infarct, and most all softenings found by me occurred on the boundary region of the different circulations. When a hemorrhage occurs it goes in a ventral direction because of that being the direction of least resistance.

The island of Reil forms a solid formation, and that with the skull as a base sends the blood in a medial direction. The island of Reil is embryologically very different from the striatum. In a two and one half months embryo given by Edinger, the striatum rises free from the base of the brain, distinctly separated by a fissure from the external wall of the lateral ventricle. Later it becomes so narrowed as to be invisible, but at the same time, shows a point of weakness here.

I found no lenticular striate vessels coming up in this region called the area of predilection. They arise more centrally in the lenticular nucleus and nourish the nucleus to this line.

In a number of cases a linear cyst had been formed in this, the external capsule, as a result of old hemorrhage.

Some scars in different parts of the brain, judging from the brown pigment contained, would indicate that a previous hemorrhage had coagulated extruded serum and been absorbed. Virchow designated these apoplectic cysts. This is plainly seen in one specimen. Hemorrhage formed the size of a marble around which is a distinct fibrous capsule. The hemorrhage is in the process of shrinking within the capsule. We do not know whether hemorrhages come from erosion of softened areas or not, but one case had a large hemorrhage connected with a scar in the external capsule, which scar or softening opened up the way for the occurrence of the hemorrhage. Small circular areas of softening about the circumference of a sclerotic vessel, as is so often found, may be the basis of some hemorrhage.

Charcot and Bouchard have amply demonstrated that miliary aneurysms are the real cause of many ruptures.

All the cases of hemorrhage in the ventricles, eight in number, which I have examined, show clearly an underlying softening in the basal nuclei with subsequent erosion through the lining of the ventricle and then a hemorrhage.

Extracted from the American Journal of the Medical Sciences, December, 1909

SPASTIC PARAPLEGIA DATING FROM CHILDHOOD (LITTLE'S DISEASE?), WITH LITTLE OR NO DEMONSTRABLE LESION IN THE PYRAMIDAL TRACTS.

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It is not the object of this paper to discuss the classification of cases of cerebral spastic palsies, or the relation which Little's disease bears to these cases; but rather to call attention to the remarkable and interesting fact, already discussed by Spiller[1] and others, that spastic paraplegia may occur with little or no demonstrable degeneration in the pyramidal tracts of the cord, and to add a case in point.

While a few cases have been reported bearing on this subject, the literature is not very extensive, and the last word has not yet been said. Although in some cases degeneration of the pyramidal tracts has been described, this has been caused either by a spinal lesion (Dejerine,[2] Spiller), a meningo-encephalitis (Sachs[3]), a sclerosis of the motor cortical areas (McMutt[4]), or a venous effusion into the meninges, causing destruction of the cortex (Muratow[5]). The cases of porencephalon and atrophy of the motor and other convolutions, without degeneration in the pyramidal tracts (Railton,[6] Ross,[7] Phillip and Cestan[8]), present a different problem; and yet more difficult of explanation are those cases in which there is little or no demonstrable lesion, either in the cortex or the motor tracts of the spinal cord, as in the cases of Spiller, Rolly,[9] Dejerine, Haushalter and Collins,[10] and in my own case to be reported herewith.

In some of the cases no true degeneration of the pyramidal tracts is found, and in these have been described: a fineness of the fibers of the pyramidal tracts (Spiller, Biswanger[11]), a scarcity of fibers (Ganghofner,[12] Gerlich[13]), incomplete myelization without diminution in the number or size of the fibers (Haushalter and Collins), agenesia or imperfect development of the pyramidal tracts (Dejerine, Spiller, Anglade, Jacquin,[14] Ganghofner, Cestan,[15] Steinlechner-Gretschischnikoff,[16] and Oddo[17]) or atrophy of the pyramidal tracts (Ganghofner, Steinlechner-Gretschischnikoff, Oppenheim[18]); while an effort has been made to connect some of these conditions with degeneration of the cortical ganglion cells, or diminished number, or absence of these (Biswanger, Rolly, Otto,[19] Köster,[20] Sachs).

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[1] University of Pennsylvania Medical Bulletin, January, 1905; Jour. Nerv. and Ment. Dis., 1898, 81.

[2] Revue neurol., 1903, xi, 601. Comp.-rend de la soc. de biol., 1897, p. 261.

[3] New York Med. Jour., 1891, v, 1.

[4] Amer. Jour. Med. Sci., 1885, 58.

[5] Deut. Ztschr. f. Nerv., 1896-97, x, 272.

[6] Brit. Med. Jour., 1892, i, 441.

[7] Brain, 1882, 473.

[8] Comp.-rend. de la soc. de biol., 1897, p. 1080.

[9] Deut. Ztschr. f. Nerv., 1901, xx, 152.

[10] Comp.-rend. de la soc. de biol., 1905, lix, 223.

This brief and hasty review of the pathology of the cases of cerebral spastic palsies dating from infancy or birth, does not explain satisfactorily at once the cause of the spasticity, and the paralysis in all the cases. Marie's theory that the contractures are due to absence of the pyramidal tracts, which normally exercise an inhibitory influence on the cells in the anterior horns, is not applicable in those cases in which little or no change can be demonstrated in the pyramidal tracts. Mya and Levi[21] believed also that the contractures in Little's disease were entirely of spinal origin, resulting from the predominance of the action of the cells of the anterior horns in agenesia of the pyramidal tracts. They believed that in the newborn the radicular cells of the spinal cord have an autonomous function, independent of the cortical cells. The physiological hypertonicity at this age is accentuated when the motor centres of the cord are not properly connected with the higher centres. Van Gehuchten,[22] however opposed this view, claiming that the question of age is of little importance, as the same spasticity is found in adults in affections of the pyramidal tracts. He believed that the contractures are an expression of an exaggeration of normal muscular tone, due to an interruption of the corticospinal fibers, with persistence of the corticopontocerebellospinal fibers, maintaining the cells of the cord under the influence of the motor cells of the cortex.

It is not difficult to understand that sparseness of fibers, or fineness of fibers, may have the effect of lowering conductibility, and in this way interfere with the inhibitory control normally exercised by the cortex over the spinal centres, the persistent action of the cells of the anterior horns thus giving rise to spastic conditions of the extremities.

One word as to the cause of the agenesia of the pyramidal tracts, the presence of poorly developed, thin fibers, and the sparseness of fibers in the pyramidal tracts, described in some of the cases. That these conditions result from a lack of the usual number of cortical ganglion cells is plausible, although extremely difficult to demonstrate satisfactorily. The view held by Van Gehuchten is also acceptable in this connection: that spasticity is due to the arrest of growth of the fibers in the pyramidal tracts, he having found, in fœtuses of seven months, that the axis cylinders are absent in the cord, but present above the anterior pyramids.

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[11] Virchow's Archiv, 1882, 427.

[12] Ztschr. f. Heilk., 1896, xvii, 203.

[13] Arch. f. Psych., 1891-92, xxiii, 201.

[14] L'encehdale, 1909, 252.

[15] Prog. Med., 1899, x, 102.

[16] Arch. f. Psych., 1886, xvii, 649.

[17] Arch. d. méd. d'enf., Paris, 1899, ii.

[18] Neurol. Centralbl., 1895, p. 130.

[19] Arch. f. Psych., 1891-92, xxiii, p. 53.

[20] Neurol. Centralbl., 1887, No. 10.

[21] Cited by Oddo.

[22] Revue neurol., 1897, 558.

How can the spasticity be explained when the pyramidal tracts are not degenerated? The presence of fibers in the pyramidal tracts presupposes the presence of intact cells in the cortex, or at least in the cerebrum. The character of the fibers and cells, then, must have some bearing upon the development of this symptom. When fibers of small caliber are called upon to conduct vigorous motor impulses, do we not meet with the same condition as when large currents of electricity are forced through wires of small caliber, that is, increased resistance, and imperfect conduction? The increased resistance offered by the small fibers to the motor impulses may prevent the impulses from reaching the cells in the spinal cord in toto, and thereby there is a cutting off, partial at least, of the influence of the higher inhibitory cortical centres. While, of course, this cannot be demonstrated, it seems a possible explanation of the spasticity in some cases.

The following case is interesting in this connection on account of the absence of gross and microscopic changes in the nervous system to account for the symptoms presented during life:

J. C., aged sixty-four years, was admitted to the Philadelphia Home for Incurables September 28, 1899. His father was killed in an accident, and his mother died as a result of a "broken bloodvessel." Three brothers are dead, two as the result of "kidney trouble;" the cause of death of the third is unknown. When a child the patient had measles, chickenpox, and "spasms." No history could be obtained as to the conditions at birth or during early infancy.

He stated that the present disease began at about five years of age, although he said, on a subsequent occasion, that the spasms in infancy had left him in his present condition. Physical examination showed a spastic condition of both legs, which remained stationary from the time of his admission (September 28, 1899) until his death (May 9, 1906). His gait was that of spastic diplegia, and he assumed a stooping attitude when walking. He assumed a perfectly erect position only with considerable difficulty, and could not maintain it for any length of time. The knee-jerks were much increased and equal. The Babinski phenomenon was marked on both sides, as was also the plantar reflex. There was no clonus present. His grasp in the right hand was good, but was slightly weaker in the left hand. He used his hands readily, and there seemed to be no loss of power or spasticity on either side. He spoke with difficulty, but this was due to stuttering, and there was no evidence of spastic speech. Mentally he was considerably below the average intelligence.

The autopsy was performed a few hours after death; the brain and spinal cord were removed and placed in formalin.

Macroscopically the brain showed an extensive effusion of blood in the pia arachnoid, extending over a large part of both hemispheres, and also at the base. There was no evidence of fracture of the skull or local trauma. Otherwise the macroscopic examination was negative.

Sections of the brain and spinal cord revealed absolutely nothing abnormal macroscopically. Portions of both paracentral lobules and the left frontal region were studied microscopically, as well as the pons and medulla and various portions of the spinal cord. The pia of the cortex showed a sparse cellular infiltration composed of connective tissue cells. The bloodvessels were somewhat thickened, and the cellular elements in the walls somewhat increased.

The cells of the cortex, stained by thionin, revealed no abnormality, although no Betz cells were found. The pons, medulla, and spinal cord, stained by the Weigert method, showed no evidence of degeneration. Sections of the spinal cord stained with hematoxylin and acid fuchsin, showed that the crossed pyramidal tracts on both sides took the stain slightly more intensely than the remaining white matter. Study of the fibers in the crossed pyramidal tracts by this stain demonstrated the presence of a number of small fibers closely packed together, and a sparseness of large well-formed fibers. There was a slight increase in the connective tissue, but at no time did this reach the degree which is seen in spinal cords in which the pyramidal tracts show secondary degeneration. The smallness of the fibers of the crossed pyramidal tracts contrasted clearly with the much larger fibers in the adjoining direct cerebellar tracts. The cross section of the spinal cord itself was normal in size. There were also present in the crossed pyramidal tracts, as well as in the posterior columns at all the levels studied, numerous corpora amylaceæ. The fineness of the nerve fibers was more apparent in the cervical and thoracic regions than in the lumbar region. By the thionin stain the ganglion cells of the anterior horns, at all the levels studied, showed a marked change. While there were present many normal cells, a number of cells were seen which were swollen, some showed marked atrophy of the pigment with excentric nucleus, while most of the cells showed increase in the yellow pigment. At some levels fresh capillary hemorrhages were present in the posterior horns, probably agonal in origin.

Summary. A man, aged seventy-one years, presented spastic paraplegia dating from early childhood. A study of the brain and spinal cord revealed nothing of importance, excepting a fineness of the fibers in the crossed pyramidal tracts.

In my opinion this case antedated the age of five years (as the patient stated), as his intelligence was below normal, and it could not be expected that at the age of sixty-four (when he was admitted) an accurate history of his early infancy could be obtained. The result of the anatomical study makes it possible that this case was one of Little's disease, although, of course, without definite history of the onset, this claim cannot be definitely made. It is certainly extraordinary that an individual could live to the age of seventy-one years, suffering for certainly the greater part of his life from spastic paraplegia, without its being possible to demonstrate more pathological change in the nervous system than the slight, and perhaps indefinite one which I have described. It seemed to confirm the theory, advanced earlier in this paper, that the fine caliber of the nerve cells offered resistance to the transmission of motor impulses, which, for their perfect conduction, require well-formed and large nerve fibers.

The three cases reported by Spiller are apparently similar to mine. In two of his cases he found practically nothing abnormal in the nervous system except a fineness of the nerve fibers of the crossed pyramidal tracts, and in the third case there was no positive microscopic lesion whatever. He believed there was an agenesia of the pyramidal tracts in the spinal cord in the first two cases cited, while in the third one the explanation offered was that the nerve fibers had not grown downward as far as the lumbar region in normal numbers, although he confessed that this was difficult to prove.

CONCUSSION OF THE BRAIN

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In this day and generation a justification is desirable when a single case is reported. This is particularly in order when the case is one of cerebral concussion. Nevertheless, on account of the extraordinary symptom-complex and course of the case in question, I offer no apology for its presentation.

History.—August 6, 1910, E. D., male, aged 6½, weight 49 pounds, in robust health, fell while running on the porch, striking the left side of his head. The time of the accident was about 4 p. m. Judging from the position of the echymosis, which was very slight, the point of impact must have been about 4 cm. above and 2 cm. behind the external canthus of the left eye.

I saw the child within ten minutes of the accident. The nurse said that after he fell he got upon his feet immediately, crying lustily. He was very pale, and the nurse thinking that he was more scared than hurt, sought to pacify him by walking. He had not gone far before he said: "I can't see anything but sky."

Examination.—My examination revealed the following condition: Total blindness in both eyes for both form and light. A lighted candle held 25 cm. from his eyes in a darkened room gave no visual impression of light. The irides reacted to light. All extraocular movements were perfectly normal. The hearing in each ear was normal. The tongue was protruded in the median line. There was no involvement of the facial muscles, and the elevation of the soft palate was normal and symmetrical. The movements of the upper and lower extremities were normal in all directions. The reflexes of the upper extremities were normal and no spasticity was present. Both knee jerks were greatly exaggerated, and although there was no ankle clonus present, yet the sudden flexion of the foot at the ankle provoked one quick reflex extension movement. Passive movement of the lower extremities demonstrated a very decided degree of spasticity. Normal plantar reflex was present on right, but a well marked Babinski reflex was elicited on the left. The pulse was full but irregular, and at the rate of 68 to 72 per minute. The breathing was deep and sighing in character, and at the rate of 16 per minute. He answered question readily, and showed great mental distress, because he was afraid he would never see again. After the examination he quickly went to sleep. He could be aroused from this sleep, and showed no predisposition to lie on either side. During this sleep the breathing still maintained the deep sighing character.

I made my second examination about one hour after the first, at which time all the symptoms as above recorded were present, with the exception that on testing the eyes with the candle he said that it seemed a little lighter when the candle was placed 25 cm. in front of his eyes. The Babinski reflex on the left at this examination was pronounced. In a half hour from this time he called a lighted candle a lighted match. In another half hour, two hours from my first examination, he was able to recognize a lighted candle as such, but could not distinguish objects, such as a pair of scissors, knife, picture, etc. From this time on his eyesight returned with great rapidity, and three hours after my first examination he was able to distinguish objects and people with great precision. On account of his general condition I did not make any examination of his visual fields at this time. His temperature never went above 99.4 F. At 10 p. m. I found his vision normal, pulse 72, temperature 98.4 F., respiration 18. At this time I found the Babinski reflex on the left hard to elicit but questionably present. The chief symptom at this time was vertigo on motion.

Subsequent Course. The next day he felt perfectly well except for the vertigo which was present when he was propped up too erectly in his bed. He had an attack of nausea and vomiting at 5 a. m. A careful examination for hemianopsia, astereognosis and lack of sense of position in the limbs proved negative. His reflexes were all normal and the Babinski reflex on the left was replaced by normal plantar flexion. The recovery was uninterrupted, and four days after the accident he was leading his normal life. There was never any disturbance of bladder or rectal functions. The only therapeutic treatment was magnesium sulphate to open the bowels.

The case is interesting on account of the rapid clearing up of the complete blindness, the sudden causation of the monolateral Babinski reflex, which cleared up within eighteen hours, and the very evident interference with the spinal reflex inhibitory mechanism with no symptoms of compression.

After writing the above report, the *Deutsche medizinische Wochenschrift* (August 4, 1910) has come to hand, in which is a short paper by Hirsch of Prag ("Ueber passagere Rindenblindheit durch Commotio cerebri"). There is no mention of any examination for involvement of the central nervous system, other than a careful examination of the eye. He cites Wildbrand and Sänger ("Die Neurologie des Auges") as having collected seven cases of similar nature.

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SCIATICA AS CAUSED BY "RHEUMATIC" MYOSITIS IN THE GLUTEAL REGION: ITS DIAGNOSIS AND TREATMENT.

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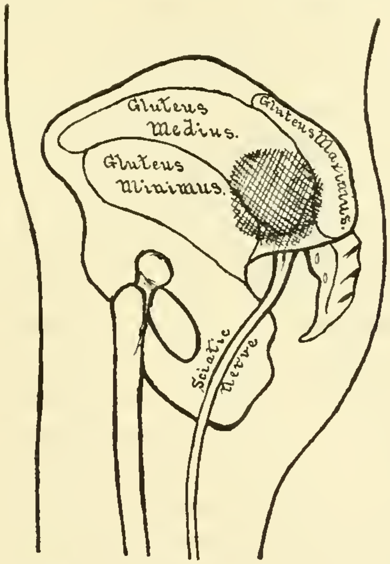
Sciatica is one of the medical terms which is used in a very broad sense. It may be due to neuritis of either the sciatic nerve or the sacral plexus; it may be caused by perineuritis; it includes neuralgia of the nerve; and finally, pelvic and spinal column lesions at times also give rise to sciatic pains. From such a conglomeration of causes and with an affection which at times is most painful, the ability to place one's finger on a certain type of cases, with the assurance of relief to the patient, is most gratifying.

The neurologist frequently sees patients who present the symptoms of pain in the gluteal region and dorsal aspect of the lower extremity and with some disability; furthermore, Lasègue's sign may be present, with impairment or loss of the Achilles jerk and also with cutaneous sensory changes. These cases are, of course, diagnosticated sciatica, but in some of them a careful examination will show an absence of tenderness along the course of the sciatic nerve. What at times is actually the cause of these symptoms is rheumatic myositis of a part of the gluteus medius, with perhaps slight involvement of the gluteus maximus and gluteus minimus muscles. This particular localization has not yet been recognized by our text-books, though it may be easily demonstrated. It appears that the only careful study of myositis in this region has been made by Professor Karl Petrén[1] of the University of Upsala, and he speaks of this group of cases as presenting "the common picture of sciatica with the usual localization of the pain but with absence of tenderness to pressure over the nerve and tenderness to pressure over a certain area of the gluteal region." It is possible that Valleix[2] had in mind this gluteal area, though his point of tenderness was not exactly at the same location. Perhaps it should be added that Bruce[3] also observed a large number of cases of sciatica, upward of 400, he states, but because of his absurd views little value has attached to his work; he did not believe there was such a condition as sciatic neuritis, but held that all cases of sciatica were due to rheumatoid coxitis.

We are constantly recognizing a larger group of manifestations resulting from auto-intoxication, which are for the most part of intestinal origin, though by no means necessarily so, as, for instance, oral sepsis. There are many nervous symptoms produced in this way, and some of the psychoses are thus caused; ultimately more or less widespread arteriosclerosis develops. Headaches from this condition are exceedingly common;[4] local areas of muscular resistance presenting marked tenderness upon pressure develop in many parts of the body. It should be remembered that any voluntary muscle may become rheumatic, that certain areas are quite prone to this invasion, and one of these sites is the gluteal region. Certain it is that many errors in diagnosis result from our lack of knowledge of these rheumatic indurations.[5] [6] One of the earliest students of this subject was Helleday,[7] a Swedish physician, who in 1876 reported 8 cases showing that lesions occur in various parts of the body, all of which he treated successfully; he said: "The object of these histories and what has gone before is to impress you with the importance of examining the musculature of many cases which present a picture other than that which you usually understand as muscular rheumatism." Gowers, Edinger, and others have also written of the importance of this subject. The recognition of the condition, as a rule, is not difficult, and as we have excellent therapeutic measures at our command, so much ignorance about this matter seems unfortunate. Some of the arthritic cases which lead to changes in the tendons, ligaments, and bones are closely allied to this condition. That there is a definite substance or substances concerned in the production of these indurations seems highly probable; during the manipulative breaking up of these areas it frequently happens that patients complain of neuralgic and rheumatic pains in different parts of the body; indeed, a solitary attack of gout seemed to be precipitated in this way in a case which I have referred to elsewhere.[8] In refrigeration and also after a strain or other injury, these indurations often develop, but this is most apt to occur in persons who are subject to more or less autointoxication; a microörganismal irritant may likewise produce them.

Persons do not die of this condition, and but little is known of the pathology of these areas. Vogel, quoted by Bum,[9] in section from rheumatic muscle, found thickening and adhesion of the neurolemma of the nerve branches supplying the muscle; a round-cell infiltration of the interstitial muscle substance has been observed, and this really is the so-called "fibrositis," a term which was probably first used in this connection by Gowers.[10] Owing to the intimate relation existing between muscle and nerve tissue, we have no means at our command to positively prove that the trouble is confined to either substance; it may be a neuromyositis, but for therapeutic purposes it is looked upon as a muscular disturbance, the minute nerve fibers being lost sight of, because of our ability to so successfully manipulate muscle tissue.



A common site of "rheumatic" myositis, which at times gives rise to sciatica.

Given sciatic symptoms, the diagnosis of this type of cases rests upon the muscular resistance offered in palpation and the pain elicited upon pressure, alone, at about the area shown in the accompanying drawing.

Petrén gives two possible explanations for the symptoms: "(1) that the symptoms may be due to a myositis followed by compression of the nerve, or (2) that the inflammation may have extended to the trunk of the great sciatic nerve or its immediate neighborhood"; and he further remarks that "each may hold good in different cases." The duration of the disease is uncertain; it may last for many months, but under proper treatment the intensity of the pain usually subsides in a few days, and in long-standing cases a few weeks may be required for the removal of the indurated area. The best plan of treatment is that giving temporary relief, which is to be followed by the removal of the cause. The acute cases should be given a saline purge at once and kept at rest in bed until there is an amelioration of the symptoms; this usually occurs soon, but atmospheric conditions sometimes act unfavorably. The part may be immobilized: however, there is no absolute need for this unless it affords relief to the patient. The drug of choice for internal use is aspirin, and a dram and a half or even more in twenty-four hours may be necessary; the usual doses given are entirely inadequate. Locally, mesotan or spirosal may be used. The most valuable means at our command for a permanent cure is massage; and just at this point it is worthy of note that the reason this condition is regarded a more hopeful one abroad is because of the different way in which the manipulation is carried out; there, the treatments are much more vigorous and are for the most part given with the thumbs; after a few treatments the amount of pressure required in some cases would astonish one not familiar with this work. Owing to the pain produced, it is desirable that the first few treatments be given gently. It is interesting to notice the gradual disappearance of resistance and the lessening of pain in these indurated areas as the patient improves. The treatments should be continued as long as the area is tender and the induration is palpable, or until it is determined that no further improvement can be accomplished, for it occasionally happens that the induration is of so long standing and of such density that it does not yield completely to treatment. Of some assistance in this work are the rapid vibrators whose power is delivered by an electric motor; this method of vibration has largely replaced what was formerly done by hand. As to the test of Lasègue, it is not only of diagnostic value, but may also be employed with advantage to stretch the sciatic nerve after the acute stage has passed; it may be done every day or so, gradually increasing the force, since too sudden stretching may result in permanent injury. Patrén gives it as his decided opinion that "Lesègue's symptom in sciatica and Kernig's symptom in meningitis are one and the same phenomenon." This may be true, since Kernig[11] has recently given us as the simplest method of establishing his symptom, having the patient endeavor to elevate his extended leg while lying on his back; a number of writers have found this to be the most satisfactory method of applying Lasègue's test. In cases of long standing and where the symptoms persist after vigorous manipulative treatment potassium iodide is of some value. Because of its escharotic and resolvent properties, thiosinamin has been considered useful; it is by preference administered hypodermically; this substance is not freely soluble, and the injection of an alcoholic solution is quite painful; when combined with sodium salicylate a double salt is formed which in trade is known as fibrolysin; this is freely soluble in water, the injection of the solution is practically painless, it is absorbed promptly and is said to be at least as efficacious as thiosinamin. It would seem that fibrolysin is worthy of a more extended use than has been accorded it in this country. Its injection may be subcutaneous, intramuscular, or an intravenous one; it acts perhaps as well when thrown directly into the muscle tissue, though not necessarily at the site of disease; after a number of injections manipulation may be begun. As supplied, each tube contains 2.3 c.c. of a 15 per cent. aqueous solution of fibrolysin, all of which should be used at once. Improvement may begin after about ten injections, but many times that number is often required. Luff[12] has used it in a variety of chronic rheumatic cases, and gives it as his decided opinion that it was more or less successful in two-thirds of the cases. Fibrolysin is contra-indicated in cases where old cicatrices might be affected, as after abdominal section, where there are chronic latent inflammations which might be lighted up as in tuberculous persons, and in the aged with arteriosclerosis.

Electric treatment of rheumatic cases seems to have no great curative value; the high frequency current has been much extolled, but the benefit seems to consist in its slight amelioration of the pain. A weak faradic current is somewhat useful after the acute stage has passed. Dry heat is grateful, and it may be applied by means of an electric extension cord, the bulb being placed over the part with a piece of cloth interposed. Moist heat should be used carefully, otherwise the patient may catch cold from rapid evaporization of moisture, thus aggravating the condition.

In persons who have these rheumatic tendencies a great deal may be done to prevent their developing into actual disease, but this can only be accomplished through persistent and rigid attention to hygiene.

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SIMULATED INSANITY*

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INTRODUCTION

One simulates insanity who, being sane, by his action and speech or by his suppression of both speech and action, tries to prove that he is of unsound mind. One who is insane may also feign the symptoms of a type of insanity from which he does not suffer. For one simply to assert that he is insane, which is occasionally done, does not constitute simulation of insanity. Such assertion must be accompanied by some evidences of the playing of the part in order to constitute feigning. One dissimulates insanity who endeavors to conceal the fact that he is insane. This pretense of sanity by the insane is a far more common procedure than the feigning of insanity by the sane.

Well-known historical instances of the feigning of insanity are to be found in the classical works on mental and forensic medicine—the feigning of Brutus in the time of the Tarquins, of Ulysses as recorded by Homer, of Solon in an effort to stimulate the Athenians to recover Salamis, and of David in the Old Testament. In each of these cases, and in other similar historical examples of simulation, the methods used to carry out the feigning were of a primitive, or at least of a comparatively simple character. They were, however, the methods which in some cases are still resorted to by the simulator, and this because the popular idea regarding insanity is not much in advance of the views held in the time of the Tarquins. They are methods based either on crude conceptions of the nature of

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mental disorder, or on the signs of such disorder as observed in two or three types of insanity. Solon's simulation of insanity was, as Dr. Lloyd[1] aptly remarks, an instance of the lawyer and not the client practicing simulation. It may be said, however, that, while the attorney does not himself feign insanity for a purpose, he has been accused, apparently with justice in some instances, of instigating such simulation in the client. Instances of such instigation are, however, extremely rare.

COMPARATIVE RARITY OF THE SIMULATION OF INSANITY BY THE SANE

Contrary to what is believed by members of the legal profession and the public at large, feigning of insanity is rare, and, above all, is rare in cases which come under observation in the private practice of medicine and in institutions for the insane. It is also comparatively rare, contrary to general belief, in cases in which the plea of insanity is entered as a defense in homicide and other cases. The most frequent instances of real simulation are observed in the military and naval service, in prisons, and in asylums or hospitals for criminal lunatics; but even in these it is not as common as is supposed. I have had no experience in the study of insanity, or of its simulation in criminal lunatic asylums, or in either the military or the naval service. My studies in penal institutions have been confined to the opportunities offered by one or two general investigations of the mental status of inmates of prisons, and to the examination of a considerable number of cases held for homicide or other serious offenses. The occurrence in prisons of insanity which is overlooked or disregarded is far more common than the simulation of insanity by prisoners.

In a recent homicide trial in which the plea of insanity was entered one of the medical witnesses for the prosecution, in response to a question by the attorney for the defense, said that he had seen about fifty cases of the simulation of insanity, mostly in the psychopathic wards of one of the hospitals in a large American city. In connection with the Philadelphia General Hospital are such psychopathic or detention wards, with which I have been connected for nearly a score of years, but, strange to say, I have never seen a single case of the feigning of insanity in those wards. Several of my colleagues, with whom I have conferred, have had the same experience.

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[1] Lloyd, James Hendrie: Insanity, Its Various Forms and Its Medicolegal Aspects, Book ii; Wharton and Stillé's Medical Jurisprudence, Vol. 1, Edition 5, 1905.

In a large private and hospital practice extending over many years I can recall only a single case in which I was convinced that genuine simulation had been attempted, this not including the homicide cases to which reference will later be made.

It may happen, of course, that in particular periods a larger number of cases of simulated insanity occur in certain institutions, a fact which accounts in measure for the difference of opinion as to the frequency of simulation in prisoners and insane hospital patients.

OBJECTS FOR WHICH INSANITY IS FEIGNED

The objects for which insanity is feigned are various, among the most important being the desire to escape military service, to exchange less for more comfortable prison quarters, to annul contracts, and to escape punishment for minor or major criminal offenses.

In recent times a number of instances have occurred of the more or less successful feigning of insanity by newspaper reporters who have sought in this way to enter and sojourn in hospitals for the insane, in order to write sensational articles about the alleged abuses in such hospitals.

In 1889 a reporter succeeded by simulation in having himself committed to the Philadelphia Hospital for the Insane, where he remained for a few days and then published a story of what he alleged he had seen and heard which produced a transient sensation. He was probably never thoroughly examined by members of the hospital staff, although he was seen by several of them during his brief stay. Some points in the case are worthy of recall, as illustrating the manner in which the intelligent but badly informed simulator overdoes the rôle. No really insane man ever indulged in the entertaining and extravagant performances of this scribe. He was picked up while creating some excitement at one of the Philadelphia street corners. He asserted that he had lived at the bottom of the sea and was 1,000,000 years old, that he had had nothing to eat for ninety years, that he was the last of the actors, that they were all killed by the song-and-dance men—killed with cannon—and the leaves of the trees had turned red with their blood.

"My home," he said, "is in a cave in a mountain on the planet Mars. It was an elephant's nest, but I made friends with him and he brings me food every day. At night I sleep under the sea in a coral grotto, while mermaids sing me soft lullabies that woo the drowsy god." He kept up this farrago or something like it for a day or two; then his identity was disclosed and he was discharged from the hospital.

THE TESTING OF SIMULATION

It is not my purpose in the present article to go at length into the question of the methods of testing simulated insanity. These are given at more or less length in works on insanity and forensic medicine. They include many devices, such as making remarks regarding the patient or his symptoms which are calculated to induce the simulator to assume that which is suggested; suddenly charging feigning and watching the effects; the use of emetics, ether, chloroform, etc. It must be said that all such methods will fail in particular instances, either through foreknowledge of what is likely to be expected or through the natural shrewdness of the individual being investigated. Setting a watch by day and night through an opening in the wall, ceiling, or door is sometimes resorted to, and has occasionally proved efficient in unmasking a feigner. It fails sometimes, one of the reasons being that occasionally unusual powers of endurance and unusual cleverness in keeping up violent and grotesque speech and action are shown by the malingerer. Tests which involve the idea of anesthesia, which the simulator is likely to believe should be present, like the others, may fail, as may also those which involve cruel procedures such as the use of sharp or heated instruments, cold shower-baths, and physical punishment.

After all, the best procedures are those which are founded on scientific principles and the use of the ordinary methods of determining the presence of disease, mental and physical, including the study of the life history of the patient, a comparison of his present with his past behavior, the investigation of the manner in which the supposed insanity has appeared, and the determination of the existence of a type of insanity with more or less elaborate but correlated phenomena. Few simulators, for instance, have the ability to combine in one picture the symptoms of a form of insanity which contains in its ensemble phenomena of exaltation, depression, and suspicion, with certain details well known to be present. The feigning of mania, complete dementia, or stupor is perhaps most frequently attempted, but simulation is often charged in cases of paranoia. Mania or dementia are usually overdone by the simulator, as in the case of the reporter to which reference has been made. The character of the crime which has been committed and the manner of its commission may mean much to a thoroughly informed alienist.

One of the most difficult of problems connected with the question of simulation is that presented by cases of alleged or real insanity, usually forms of paranoia, or alcoholism, which present or appear to present only one or two delusions, such as those of marital infidelity and of poisoning, and yet such cases are among the most important in forensic medicine. In such cases the authentic history of the patient over a considerable period is of special value.

Mistakes are sometimes made by physicians, as well as by laymen, through not properly interpreting phenomena which appear to point to feigning. One of these not often dwelt on is that of supposing that acting, more or less consummate, is a proof of sanity. The putting of "an antic disposition on" is by no means always a proof of feigning. In katatonia, histrionism or theatrical behavior is frequently present. The poses, forced gait and declamatory gestures exhibited by a Chinaman recently tried in Philadelphia apparently did much to prejudice those who examined him, as well as the jury to whom his behavior was described, against the opinion that he was insane, and yet this conduct was one of the real evidences of his mental disorder.

THE DISSIMULATION OF INSANITY BY THE INSANE

The simulation—or, it would perhaps be better to say, the dissimulation or concealment—of insanity by the insane is comparatively frequent. The attempts at concealment are made for a variety of reasons—to escape from institutions by deceiving officials or visitors; to obtain discharge on a writ of habeas corpus;

to arouse sympathy; to prevent certification, or the appointment of a committee; or to obtain certificates of sanity to be used as instruments of defense or aggression. Many such cases have come under my observation. In some instances the concealment of insanity has been so successfully practiced as to deceive not only laymen and legal advisers, but honest and capable physicians.

The insane who try to conceal their mental disorder sometimes have advantages over those who, being of a sound mind, simulate insanity. When the former have been inmates of hospitals for the insane, and are intelligent and observant, they have the opportunity not only of studying the phenomena of alienation as exhibited by others, but of learning through the examinations made by physicians what symptoms and signs are regarded as most significant of the presence of insanity. If they have sufficient mental power, profiting by the knowledge thus obtained, they are able (usually only for a time) to conceal the evidence of insanity in their own persons. When not inmates of institutions, they may in some cases, by simply closing their mouths and controlling their conduct, be able to keep in check or out of sight insane manifestations. It requires in some cases unusual skill and patience to elicit the evidence of insanity, and it may indeed be impossible to do this at some particular examination. Perseverance, however, will usually be rewarded by success.

Every neurologist or alienist of considerable practice is now and then asked to give a certificate of sanity, a request which in itself should incline him to be on his guard. In a few instances I have known unwary, although honest, physicians to give such certificates, which they subsequently have had cause to regret.

A woman who made the lives of some of her townsmen, medical and otherwise, and of at least one man of eminence in public life, more or less intolerable by her accusations and undesired attentions came to me from a distant state, as she had gone to others in more states than one, to obtain a certificate that she was sane. She was extremely persistent in her efforts. She was a woman of good presence and some ability, or at least she had superficial acquirements which she used to advantage in the pursuit of her purpose. It was only by a close study of the history of her case and of her statements and personal bearing that a conclusion was reached after several interviews. She undoubtedly belonged to that numerous class included under the broad term of paranoiacs, with delusions both of self-importance and of persecution, the former at the time of my examinations predominating. Her egotism amounted to a megalomania, and close investigation showed that she might become dangerous under provocation or indifference. My refusal, after careful study, to give the certificate desired freed me from her personal visits, but not from her abusive attentions by mail.

In another case—that of a man who had made his escape from an asylum in a neighboring state and was for a time in self-imposed seclusion—refusal after prolonged investigation to come over to the side of the accused again brought on me maledictions. This man had several interesting forensic campaigns in which the question of his sanity or insanity was the chief object of conflict.

A woman 35 or 40 years of age came to my office, accompanied by an intelligent young man who had espoused the cause of her sanity, as appeared later, and requested me to give her a most searching examination and tell her if I could find in her any signs of insanity. She was agreeable in her demeanor and apparently honest in her desire to arrive at the truth. Beginning with her earliest history, so far as could be learned from her statements, and continuing by methods of examination and cross-examination, I tried to penetrate the armor of deception with which she had encompassed herself. It was a long task and not an easy one, but after nearly two hours I determined to my own satisfaction at least that she was not of sound mind, and then learned that she had but recently been allowed to leave a hospital for the insane in which she had been confined for many months. In spite of her skilful resistance a delusional state was uncovered—one which had brought on herself and others much and somewhat serious trouble. Her concealment of her insanity was a more skilful piece of sustained acting than has ever come under my observation in any case of alleged simulation of insanity.

Another case, one of the most positive examples of dangerous paranoia which has come under my observation, was certified by another physician and myself to a hospital for the insane. He had hallucinations of hearing and of sight, delusions of persecution by unseen agencies such as electricity conveyed by wires, and other of the classical phenomena of this form of insanity. He had also well-marked delusions of self-importance, and had had episodes of excitement in which he threatened to kill his wife and others. After his admission to the hospitals he resisted all efforts on the part of the asylum physicians to demonstrate his delusional state. A writ of habeas corpus was asked for, and several eminent alienists and neurologists examined him with negative results. They could not obtain evidence which satisfied them that he was laboring under the delusions and hallucinations which had led my colleague and myself to certify him. The case was heard before one of the most conservative of our Philadelphia judges, and on the testimony produced at the hearing the man was remanded to the hospital for further observation. He still successfully continued his policy of suppression, and was some weeks later brought into court and this time discharged. His later history was interesting and somewhat dramatic. His delusions after he was set at liberty again became apparent. He made many threats and several assaults, and after some difficulty he was again certified and sent to one of the state hospitals, where he still was a short time since, his insanity being almost continuously in evidence. It was only by the most fortunate chance that this man did not kill some of those who were the objects of his insane suspicions.

Paranoia is not the only form of insanity which is occasionally successfully suppressed or abates in such a way as to deceive those usually capable of determining its presence.

A few years since, on the certificate of another physician of Philadelphia and myself, a paretic patient was sent to one of our best known hospitals for the insane. Partly by personal suppression and partly, no doubt, under the influence of conditions which bring about marked remission in symptoms, this man became so quiet and self-controlled as to deceive a physician of some distinction, and also apparently some of the physicians of the hospital, into the belief that he was either not insane or had recovered. He obtained his discharge from the hospital, and immediately went about securing the services of attorneys to bring suit against myself and others for conspiracy in having him placed in the institution. While some attorneys refused, he succeeded in obtaining the services of one or two, and had carried matters so far that suit was about to be entered when his insanity became so apparent, even to those who were helping in his crusade, that all concerned were placed in an unpleasant predicament. Eventually he was returned to the hospital.

SIMULATION IN HOMICIDE CASES IN WHICH THE PLEA OF INSANITY IS OFFERED

In order to recall some facts of value from experience I have collected the data of twenty-four cases in which in trials for homicide the plea of insanity was entered as a defense. In these cases I appeared seventeen times as a witness for the defense; three times as witness for the prosecution; as special adviser three times, and once as arbiter, chosen by agreement of defense, prosecution and the court. By adviser is meant that I was called on to counsel with the attorneys either for the defense or prosecution; for the former in two instances and the latter in one. Simulation was charged or inferred by the prosecution in twelve cases; in two cases the plea of insanity was accepted with little or no contest. It might be said in passing that in both of these instances the accused were women. Ten cases were tried on their general merits with little or no reference to the question of simulation; that is, it was simply held by the prosecution that the accused were not insane, and by the defense that they were.

Recalling these cases after many years, my judgment has but little changed from the time when, full of interest in the details of the investigation and in the events of the trial, I held to my conviction as to the sanity or insanity of those under trial or about to be placed on trial.

In only one case was I impressed with any considerable doubt as to the validity of the insanity plea, and in this case the man himself constantly asserted that he was not insane. It was indeed not held that he was, except at the time of the commission of the crime for which he stood accused, and which was not denied by him or his counsel.

The case was one in which the evidence seemed to point to transient insanity in an epileptic—a most difficult plea to uphold, and yet one than which there is none more likely to be just if the history of the case affords sufficient proof. The man had an attack of spasm apparently with unconsciousness in the court. I was not present at the time and had not the opportunity of studying his condition during the seizure, but a physician of ability, although one not an alienist or neurologist. believed that the attack was genuine. The accused had on his head the scar of an old injury. pressure on which caused pain and brought on a vertiginous state.

Of the seven cases in my list in which there was acquittal on the plea of insanity in only two was there any possibility of simulation, and in both of these cases this might be regarded as doubtful. One of the accused, however. was a highly educated man and had the ability and probably the knowledge of mental disorders necessary to aid him in his purpose if he designed to feign. The other was an ignorant but rather shrewd man, who might have made a rude attempt at simulation. In the first case the evidence as to the existence of melancholia was sufficiently decisive, and in the other acquittal was obtained from the standpoint that the man was suffering from a form of delusional and hallucinatory insanity with amnesia.

As has already been indicated, three of those acquitted were women, and sex probably had as much as anything else to do with the rendering of the verdicts. In two of these acquittal was practically ordered by the judge with the acquiescence of the prosecuting attorneys, and I have no doubt that in both instances the best interests of justice were furthered by the action. In the other case the woman was acquitted in spite of vigorous resistance on the part of the prosecutor. I was not retained as a witness in this trial, but was familiar with the details of the case, and was employed to give an opinion some time after the conclusion of the trial as to whether she should be released from a hospital for the insane to which she had been sent after her acquittal. The cases were fought out mainly on other lines than simulation.

Without doubt the feeling in the minds of judges, juries and the public is against the infliction of capital punishment on women, although at times this feeling is overridden in the interests of justice or for other reasons not always apparent.

In another case after a careful examination of the prisoner and a careful going over of the history of his case and the testimony, I came to the conclusion that the interests of the defense would be best served by not calling on experts, rather trusting to lay and ordinary medical evidence and to the eloquence of the attorneys. The man did not make the slightest attempt at simulation, at least not at the time of my examination. He seemed careless of results and not desirous of being regarded as insane.

In the seventh case the testimony was conflicting, but the accused was sent to an asylum as the result of an agreement to have the case arbitrated by some one who would have no connection with the trial or the investigations leading to it. Feigning, so far as I know, was not attempted.

Several years since with Dr. E. N. Brush, now superintendent of the Sheppard and Enoch Pratt Asylum, at Towson, Maryland, I was employed as a witness in the case of a German who had committed homicide. After this man's execution his brain was secured by Dr. Brush, who made a report, not only on the case in its general features, but on the findings of the necropsy. The brain was extensively diseased. The lateral aspect of the right hemicerebrum showed a marked porencephaly. There was arrested development in the left half of the body, the patient having been asymmetrical from birth. His skull was asymmetrical. He had the history of having a fall, was intemperate, and had had a sunstroke. His knee-jerks were absent, his pupils unequal, and his symptoms and the postmortem findings pointed to general paresis in addition to the congenital or infantile arrest. He was 57 years old at the time of the trial.[2]

It is a matter worthy of passing notice that one of the chief elements which appeared to bring about the conviction of this man was the use which the prosecuting attorney made of the excitement with which the country was then filled about anarchy and anarchists. The trial was held a short time after the Haymarket crime in Chicago, at which time a number of police were killed or wounded as the result of bombs alleged to have been thrown by anarchists, several of whom were afterward tried, convicted and executed for inciting the crime. The prisoner was a German, and this with the homicide itself was sufficient to make him an anarchist and anarchy responsible for his deed.

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[2] Brush, E. N.: Polyclinic, Philadelphia, vol. 5, 1888.

The only woman whom I remember to have been executed in Philadelphia during my professional life was a weak-minded and probably irresponsible creature, although not in a technical sense insane—not one whom physicians would have been likely to certify to an institution after the usual examinations. This woman had not sufficient mental power to feign with success anything that required the exercise of any considerable degree of intelligence. She poisoned several of her children by administering arsenic to them in their food, this crime seemingly having been done for the purpose of collecting a small insurance on their lives.

An early case in my experience was that of a man who had killed his mother-in-law and wounded his wife. He was supposed to be suffering, among other things, from delusions of marital infidelity. These, as every experienced alienist knows, are among the most dangerous of all delusions. They are sometimes associated with few other evidences of insanity; they occur in cases both of alcoholism and other drug habits and in constitutional paranoia. They are likely to be looked at askance by judges and by the community as represented by juries. One insane in this way is never likely to admit his insanity. Four men out of five appointed by the governor to inquire into the sanity of the accused reported him as insane, but the governor refused to pardon him and he was executed. That simulation was not practiced in this case was my judgment and that of my colleagues, although this may not have been held by others.

On Oct. 17, 1887, was begun one of the most interesting trials for homicide in the annals of Philadelphia jurisprudence. Oscar Hugo Webber, on Dec. 6, 1886, without provocation, killed William Martin, a jeweler. Webber, about a week previous to the shooting, had taken a clock to Martin to be repaired. When the work was done he took the clock home, but about a week later brought it back. He and the jeweler had some words, when the former drew a pistol and fired four shots, killing the latter. Webber's father was insane, and one of his sisters was an epileptic. With Dr. James Hendrie Lloyd I examined him for the attorneys who had been appointed by the court to defend him, visiting him five times in all. I became thoroughly convinced of his insanity.

At the trial a considerable number of witnesses, both medical and lay, testified to his delusions and insane acts. One physician, who had known him for ten years and attended him and other members of his family, had some time before the homicide recommended that he be placed in a hospital. The accused believed that his wife was unfaithful to him, and wished to drug him to get him out of the way, and that his children were illegitimate. He also believed that he was the victim of persecution of his friends, relatives and fellow workmen, and even those with whom he came only occasionally in contact. No clearer case of insanity was ever admitted to a hospital for the insane.

No medical evidence was offered in rebuttal, although several lay witnesses testified in rebuttal their belief that he was sane. It is to be inferred that the prison and other physicians who are known to have examined him in prison before the trial, had reported to the district attorney their belief in his insanity. Whatever is the truth regarding this, it remains that they were called neither by the prosecution nor by the defense. Webber was convicted and sentenced to death. Evidently, however, some force was at work which withheld the hand of the law in the final act. He remained in prison until he died, becoming more and more delusional and demented.

Dr. Lloyd[3] has written an interesting article regarding this case.

In this case an effort was made to have proceedings instituted to determine, first, the extent of sanity or insanity of the accused, but it was decided that the trial should go on in the usual manner. As Lloyd remarks, although the law authorizing the procedure would appear to be one adapted to furthering the ends both of mercy and of justice, it is seldom invoked in the defense of those homicides alleged to be insane.

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[3] Lloyd, James Hendrie: The Insanity of Oscar Hugo Webber, Proceedings Med. Jurisprudence Soc. of Philadelphia, Nov. 13, 1888.

The case was relentlessly pressed to conviction. Simulation was not attempted by the accused. When the man rose to plead, instead of leaving this duty to his counsel, as is usually done, he stammered some incoherent words. When he was ordered to sit down, the judge exclaimed, "We will have no Guiteau business here." On one or two occasions when Webber was examined, efforts were made to get him to give some reason for the killing of Martin, who had not injured him in any way, but the accused said he would tell all about it when he got to court. He seemed to have some insane idea that he would exculpate himself by telling the court why he had done the deed. It is doubtful whether he had sufficient mental power to carry out even the feeblest effort at feigning, if such an idea had ever entered his mind.

A homicide of great interest and one in which simulation was charged was that of Joseph Taylor, who in October, 1884, was convicted of murder in the first degree, he having killed a prison keeper. Taylor was a man of low type, a so-called social pariah, who had been arrested and convicted of numerous crimes. His brain was an interesting teratologic study, and was one of several criminal brains described by me in the presidential address[4] before the American Neurological Association in 1886. One hemisphere was distinctly different from the other in bulk and height, and the lobes, gyres and fissures presented many fetal and ape-like similarities. It was a low-type, arrested, aberrant human brain in the fullest meaning of these words.

The conclusions reached by the medical investigators and witnesses for the defense was that Taylor was a delusional monomaniac of the so-called criminal or prison type. He was, so we believed, the victim of well-marked delusions of persecution, these being more or less connected or correlated, and having reference to the putting of injurious or poisonous medicines into his food and drink, and to bad treatment from the warden, keepers, doctors and prison officials generally. Numerous other factors entered into the delusional state of the accused, but it is not in the scope of this paper to discuss this or any other case in much detail.

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[4] Mills, Charles K.: Arrested and Aberrant Development of Fissures and Gyres in the Brains of Paranoiacs, Criminals, Idiots and Negroes: Preliminary Study of a Chinese Brain, Jour. Nerv. and Ment. Dis., 1886, xiii, Nos. 9 and 10.

The question of simulation was made an important one in the course of this trial. Testimony of the prison physicians was produced which showed that he had told the doctor several times before the trial that there was nothing in the crank business and that he was going to work. On the first day of the trial Taylor had several outbreaks in the court, which impressed some with the idea of feigning, and others that they were somewhat additional evidences of his insanity. Whether Taylor did or did not simulate in prison and in court, there never was any question in my mind as to his insanity. His symptoms were in accord with a certain type of insanity which he had neither the experience nor the mental power to feign successfully. It can not too often be insisted on that a lunatic, like a sane man, may fuss, or feign, or fight, or lie, or, in short, may commit any offense against righteousness or the law. The existence of sanity or insanity must be decided altogether independently of any considerations as to the moral obliquity of the individual, except in so far as this directly elucidates his insanity.

About eleven years ago in one of the interior cities of Pennsylvania a man was tried, convicted and executed for shooting another man who had attempted to enter his house to serve a writ, or in some way to carry out some form of legal process. The man who committed the homicide had barricaded himself, somewhat after the fashion of the sixteenth or the seventeenth century or earlier, as detailed by more than one novelist. This man's family and past history, the extraordinary views which he held as to his rights and wrongs, the manner in which he committed the crime, his conduct before, at the time, and after the killing were among the evidences of his probable insanity. Many things in his history pointed to a long-existent paranoid state with maniacal episodes.

Feigning was asserted in this case, and there was some evidence to support the idea that the accused had shammed some things during his confinement. Simulation, whether present or not, was not inconsistent with the form of insanity from which he was supposed to be suffering.

Like Taylor, he gave a dramatic exhibition in court which inclined some of those who observed it to the view that it was feigned, and others that it was simply the impulsive act of a man of unbalanced mind under great excitement. While his son, a young boy, was testifying, he suddenly jumped up, and before those about him realized what he was doing ran toward the witness-box, calling out the name of his son. The latter ran as his father approached, and an interesting scene was presented of a chase of a boy by a man and of a man by the officers who soon caught and restrained him. Apparently the incident was not premeditated, but the result of a sudden impulse.

In the case of a Chinaman recently tried in Philadelphia and convicted of murder in the first degree, the question of simulation played a most important part. It was urged by the medical witnesses for the prosecution and combated by those for the defense. As this paper has already too much expanded, and as this case has not yet been heard on appeal, it is perhaps not desirable to discuss it at length. I may take the opportunity at some future time of presenting it more fully. A mere statement, however, of a few of the facts in the case will be of interest in our discussion. The accused was only about 18 or 19 years of age and had shot a man whom he scarcely knew, and with whom he had had no quarrel. In our discussion of simulated insanity the case was especially interesting with regard to the confusion of theatrical behavior with feigning; reference to this point has already been made. The case was also one in which careful investigation indicated that the type of insanity present was such as could not have been successfully feigned by any one who had not a considerable knowledge of psychiatry.

In the examinations made by the witnesses for the defense in this case the prisoner again and again said that he heard voices, that the voices told him to kill the man whom he had shot, or that he would be killed himself. He declared over and over again that the voices told him that he was the greatest of all Chinamen and that he was the prophet of Confucius. These assertions were frequently repeated in a most emphatic and sometimes a dramatic way. He arose from his seat in great excitement on several occasions, declaiming the statements with violent gesticulations.

The accused regarded himself as not only a great man, but also as one who was persecuted; as a sort of martyr and hero. He strutted into the room and around it in an extraordinary manner, with his legs drawn together or partly crossed.

A word or two might be said about those cases in which verdicts of a second degree or of some degree of manslaughter are brought. Such findings are usually in the nature of what might be termed compromised verdicts. Juries which are doubtful as to sanity or insanity, or which desire for other reasons, as because of the so-called unwritten law, bring in such verdicts when often it is quite clear that these should either be one of murder in the first degree or of acquittal on the ground of insanity. In nearly all such cases the plea of insanity is set up because the law does not seem to provide any other.

In one case in which I was a witness many years ago, the jury brought in the ridiculous verdict of involuntary manslaughter in the case of a man who had with premeditation shot and killed another man who had had illicit relations with his wife while he was away from home on business. Although the plea of insanity was entered in this case, nobody in the court room nor in the community seemed to worry much either about the plea or the verdict. The man on trial did not feign insanity, nor was it necessary for him to feign this or anything else.

A second-degree verdict was reached in the case of a man who shot and killed another man whom he believed to be the paramour of his wife, and also shot and wounded his wife. This man did not simulate insanity, but seemed to regard the plea with contempt. Nevertheless, there were facts in the case which pointed decidedly to the view that he had delusions of marital infidelity.

In another case, a young woman who had been for a short time a patient of mine was tried for shooting and killing a man with whom she had become in some way entangled. This young woman, whose people were respectable and honorable, had been a source of great anxiety and distress to her father and the rest of her family from her childhood. She was wayward, seemed to have no moral sense, and committed many outrages against propriety and even decency; she was at times depressed and apathetic and at others irritable and excited. She had not, however, presented signs of insanity which could be readily marshalled in her defense. My counsel, both to her father and to her attorneys, was not to employ experts and so bring on a conflict between the district attorney and those who were employed to defend her, but to endeavor, if possible, to get a second-degree verdict. Whether as a result of this counsel or not, this course was followed, and practically by agreement a second-degree verdict was given and she was sent to prison for many years. In this case there was, so far as I know, not the slightest attempt at feigning, but rather a condition of stolid indifference on the part of the accused.

Recently I was called on by the prosecutor of pleas, with the approval of the judge, in a city not far from my place of residence, to examine with another physician into the mental state of a man who, while in a state of drunkenness or temporary mania or both, had shot his father and mother, one of whom had died. The man had been in prison for several months, was perfectly sane, and in a comparatively good physical condition at the time of examination. His manner was frank, he did not make the slightest effort to simulate insanity, and, as was learned from the prison officials, he had made no such attempt during his incarceration, and had been in every respect a well-behaved prisoner. The only light on which to act in reporting on this, as on some other cases, was that which could be obtained from the life history of the accused man. This pointed clearly to chronic alcoholism of a serious sort. He had begun to use alcoholic beverages when a child, and as years progressed had resorted to them, with increasing frequency, changing from the lighter forms of beverage to those of a stronger kind. He had had episodes or periods of excitement and probably had had hallucinations of sight and hearing at some of these times. These and other facts led my colleague and myself to the opinion that at the time of the motiveless shooting he was probably suffering from temporary mental disorder and was not fully cognizant of the act or of its consequences. A second-degree verdict was given and the man was sent to prison for life. All sensational elements were wisely eliminated from this case by those who conducted it, its management affording a good example of the manner in which such cases should be investigated.

In only five or six of the cases thus hastily analyzed and reviewed in which simulation was alleged or inferred was there in my judgment any evidence supporting this idea. In two of these cases the individuals were really insane, but indulged in violent behavior which may or may not have had an element of shamming in it. In three of the cases the simulation, although suspected, was certainly not demonstrated.

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RESECTION OF THE POSTERIOR SPINAL ROOTS FOR THE RELIEF OF SPASTICITY.[1]

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AND

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Remarks by Dr. Spiller.

For some years I have been desirous of having the posterior roots resected for the relief of spasticity, and in a paper read before the New York Neurological Society, March 7, 1905, and published in the *Journal of Nervous and Mental Disease*, May, 1905, I advocated this operation in the following words:

"Where athetosis exists the muscles are in a state of greatly increased tonicity and often are abnormally well developed and abnormally strong. Theoretically, the proper procedure might be to cut posterior roots of the affected limbs, the number to be cut depending on the condition in each case; but this is always a serious operation, and the results have at times been unexpectedly grave."

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[1] The patient described in this paper was presented at a joint meeting of the Philadelphia and New York Neurological Societies, held December 18, 1909.

I am not aware that the operation of resecting posterior roots for the relief of spasticity had been suggested previous to the publication of this paper. Since 1905 Dr. Frazier and I have frequently discussed this operation, but we have been somewhat reluctant to undertake it.

Posterior roots have been cut to relieve pain, and in some instances serious complications have followed. It may be that some of these are the result of division of the small arteries accompanying the posterior roots, and magnifying glasses worn by the operator may allow these vessels to be kept intact when the roots are resected.

Diseases of the spinal cord have taught us that when the pyramidal tracts are degenerated, provided the cells of the anterior horns are not seriously affected, spasticity and weakness are the result; and that in some instances the spasticity is far greater than the weakness. If the degeneration of these tracts be slight, spasticity may be great and prevent motion. Physicians frequently consider spasticity and weakness as of equal importance from degeneration of the pyramidal tracts, forgetting that moderate pressure upon the cord or slight pyramidal tract degeneration may cause a high degree of spasticity and a slight degree of weakness. The spasticity often masks a considerable degree of motor power.

We have likewise been taught by disease of the spinal cord that degeneration of the posterior roots, as in tabes, causes hypotonia, sometimes of an intense degree. In posterolateral sclerosis of the spinal cord we occasionally observe a spasticity, caused by degeneration of the crossed pyramidal tracts, yield to hypotonicity as the posterior columns become affected. It was with these observations in mind that in 1905 I suggested an operation which I believed might be of service.

In cutting the posterior roots for spasticity one produces an artificial tabes. The Argyll-Robertson pupil does not develop, but hypotonia, ataxia, loss of tendon reflexes, and shooting pains are produced. Most of these symptoms are transitory. The shooting pains may be very severe at first, and are from impulses transmitted through the central portions of the cut roots, and in less degree from the handling of posterior roots that are not cut. After degeneration of the cut posterior roots has become complete the pain is likely to disappear. Ataxia is not a pronounced nor a permanent symptom. The tendon reflexes may return, although they may be of diminished intensity, and the hypotonicity will depend on the number of roots cut. By proper estimation it is possible to make the tonicity nearly of the normal degree.

The operation is permissible in almost every case where spasticity is great and motor weakness is slight, provided the condition of the patient justified it, whether the lesion be spinal or cerebral, unilateral or bilateral. It is useless where motor palsy is great.

In view of the excellent papers that have been written on this subject by O. Förster,[1] it seems unnecessary to undertake an exhaustive consideration of the operation.

The history of the patient upon whom this operation has been performed is as follows: G. K., white man, aged forty-three years, was admitted to my service at the Philadelphia General Hospital, January 6, 1907, and at the time of operation was in the service of Dr. Charles S. Potts, to whom we are indebted for the case.

On Christmas, 1903, he became intoxicated and went to sleep on the floor in a cold room. He awoke feeling cold and chilly and uncertain on his legs. For two months after this he felt weak in his legs when walking a beam, or in a perilous position—his occupation being structural iron work. On April 17, 1904, he fell backward in a dark room, a distance of one step. In falling his head struck against the wall. From the effects of this blow he became momentarily unconscious, but soon regained consciousness and tried to rise. He was completely powerless to move the lower limbs in the slightest degree. All parts below the costal margins felt as though "ants were crawling all over." He was carried to bed and slept four hours. On awakening, his condition remained as described above, the lower limbs being completely paralyzed. He attempted to pull himself out of bed, when a sharp pain was felt in the lumbar region. For ten days after the accident he retained control over his bowels and bladder, and then for eight or nine months he had to be catheterized regularly and given rectal injections to empty the bowels.

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[1] Zeitschrift f. Orthopädische Chirurgie, 1908, xxii, p. 203; and Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie, 1909, xx, No. 3, p. 493.

After nine months of catheterization he had recovered control of urination, although sometimes as much as five minutes were required in which to start the stream.

After nine months of rectal injections he could defecate, provided purgatives were taken regularly. He continued to take purgatives until his bowels were emptied normally.

From the time of the accident he was in bed nine months. He used a rolling chair about one year.

From that time on he could bear his weight on his lower limbs and moved about with the aid of a cane and one crutch, but would lose his balance trying to walk with two crutches. He states that during the first nine months following the injury he had no sensation of any kind in the lower limbs and trunk to a line about two inches above the umbilicus. Since that time sensation has returned, and he has sensation in all parts of the lower limbs.

The man was examined by me September 25, 1909, in view of operation. The notes of this examination are as follows: Voluntary power in lower limbs is about normal, but spasticity in these limbs is extreme. He cannot separate the thighs because of this great spasticity. The patellar tendon reflex is so exaggerated on each side that a tap on the patellar tendon throws the corresponding limb into a general clonus. Patellar clonus, so-called spinal epilepsy,

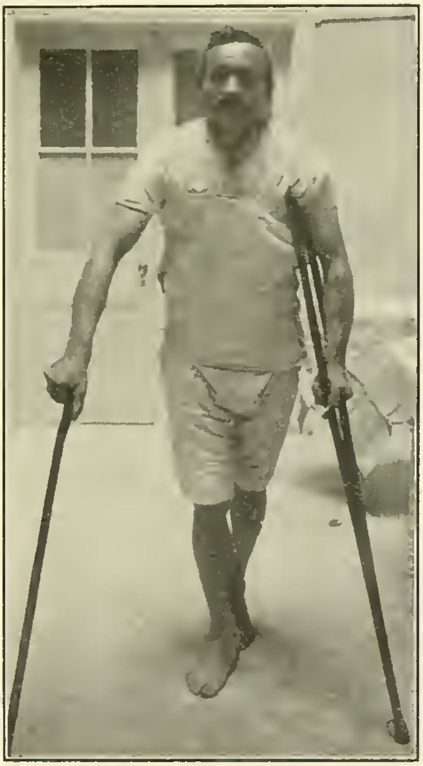

Fig. 1.—Photograph taken before the operation. Neither heel could be placed upon the ground, and the toes of one foot were brought with great difficulty from behind the heel of the other foot.

persistent ankle clonus, and Babinski reflex are present on each side. Touch, pain, and temperature sensations are normal in each lower limb. The cremasteric reflex is prompt on each side. The sense of position is normal in each great toe. He has control over the bladder and rectum. He is unable to walk without support (canes) because of the extreme spasticity; the toes of one foot catch behind the heel of the other foot in walking, and when he is standing still the heels do not touch the ground because of the spasticity of the calf muscles (Figs. 1 and 2).

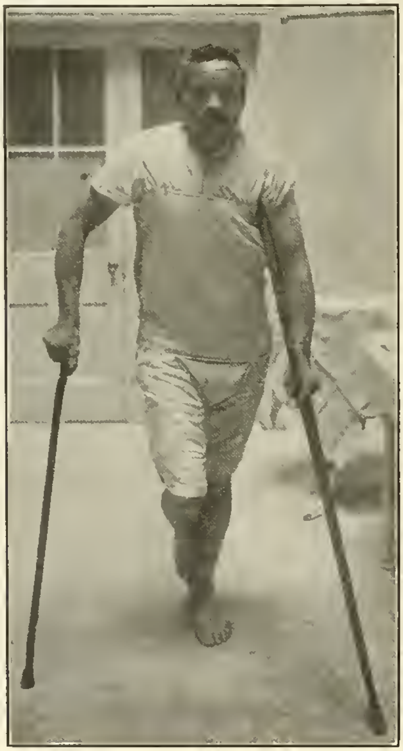

Fig. 2.—Photograph taken before the operation. Neither heel could be placed upon the ground, and the toes of one foot were brought with great difficulty from behind the heel of the other foot.

I examined the patient again on September 28, 1909, after the operation.[1] The notes of this examination are as follows: He has touch, pain, and temperature sensations in the legs below the knees and in the back of the thighs, but has not touch and temperature sensations on the front of either thigh. On the front of the left thigh he feels pin pricks, but cannot feel it on the front of the right thigh. The patellar reflex is completely lost on each side; Achilles reflex is nearly normal on the left side, but is weak on the right side. He lies with thighs far apart and legs extended. He feels touch, pain, and temperature sensations in front of the abdomen. He complains of much pain low in the back, and pain is intense here on voluntary or passive movements of the lower limbs. The cremaster

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[1] Description of operation, page 13.

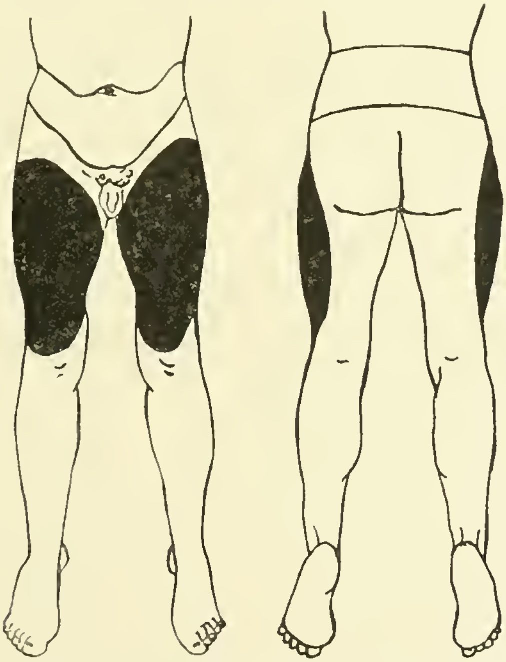

Fig. 3.—Showing the area of disturbed sensation in each thigh following the operation. It corresponds closely with the diagrams given by Seiffer (Fig. 4), but differed considerably from the diagrams of some other authors. The sensation in the distribution of the first and fifth lumbar roots was not altered.

reflex is lost on each side. He can flex slightly each lower limb at the hip, knee, and ankle, and moves his toes freely.

He moved his right lower limb freely, he says, a couple of hours after the operation, the left not so well. Both lower limbs and lower abdomen as far as the umbilicus feel as though dead.

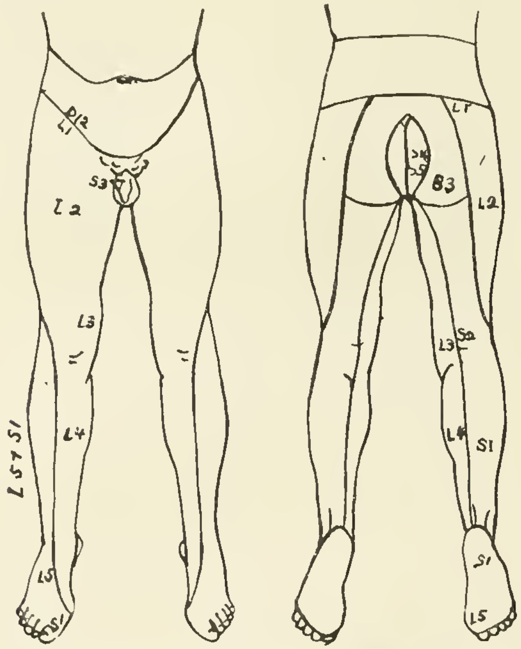

Fig. 4.—Showing the area of disturbed sensation in each thigh following the operation. It corresponds closely with the diagrams given by Seiffer (Fig. 4), but differed considerably from the diagrams of some other authors. The sensation in the distribution of the first and fifth lumbar roots was not altered.

When I examined him October 19, 1909, he was able to stand if supported, but could not put much weight on the lower limbs. Pain was not so severe now as it was soon after the operation. The movements of the lower limbs were free, and he had control of the bladder and rectum.

My notes taken December 17 are as follows: There is great impairment if not complete loss of sensations for touch, pain, heat, and cold in an area very nearly similar on the two sides and confined to the front and sides of the thighs.

This area extends, for all qualities of sensation, about equally, very nearly to Poupart's ligament in its upper limit, and to the knee in its lower limit. It extends well to the lateral portion of each thigh and well to the median portion of each thigh, while on the left thigh the sensory disturbance is one of diminution and not loss, it is complete loss in the right thigh, and the anesthetic area shades off gradually into the sensitive region. He can separate his thighs, flex and extend them to the normal degree. The patellar reflex is present on the left side in so far as there is distinct contraction of the quadriceps muscle, but there is no movement of the leg on the thigh in this reflex. The patellar reflex on the right side is present but very weak. The Achilles reflex is nearly normal on the left side, but is very weak on the right side. Typical Babinski reflex is obtained on each side.

He gets in and out of bed without difficulty. He moves his feet at the ankles with much freedom, but not to the normal degree, and places his feet firmly on the ground.

There is no positive disturbance of sensation below the knees.

The functions of bladder and bowels are normal. He goes up stairs with freedom, but has difficulty in descending stairs.

He walks with a cane and a crutch. He has still some tendency to drag the toes, but in nothing like the degree that was present before the operation. The movements at hips and knees are nearly normal, but are still impaired at the ankles.

There are several features of great interest in this case. To one who was familiar with the man's condition before the operation the results are indeed striking. The man could not separate his thighs voluntarily, nor could the examiner separate them. After the operation, when he was in bed, the thighs were widely separated, and now can be separated voluntarily in a normal manner (Fig. 5). He flexes the thighs on the abdomen and the legs on the thighs (Fig. 6) in a normal degree, and previous to the operation these movements were very difficult and almost impossible. In walking every step was made with great exertion, and it was exceedingly difficult for him to get the toes of one foot from behind the heel of the other foot (Figs. 1 and 2) because of the spasticity, and the heels never touched

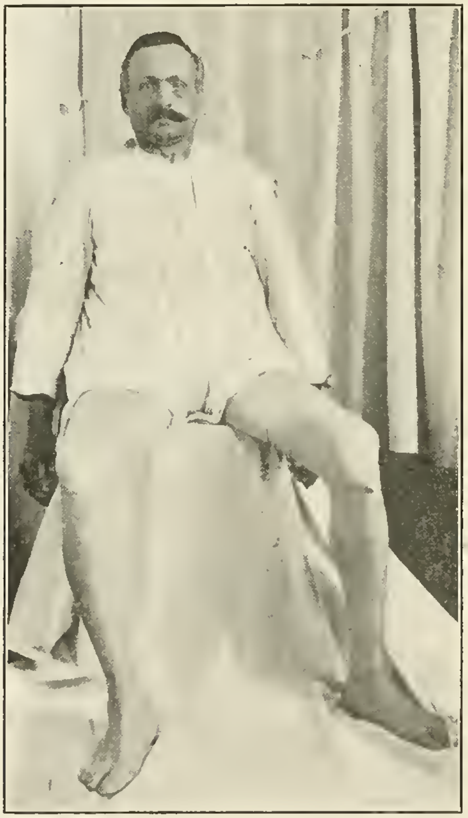

Fig. 5.—Showing voluntary abduction of the thighs after the operation.

the ground. The movements at the ankles are still less perfect than those elsewhere in the lower limbs, as only lumbar posterior roots were cut, but he places the whole foot of each limb firmly on the ground, the heel touching the ground as well as the toes (Fig. 7).

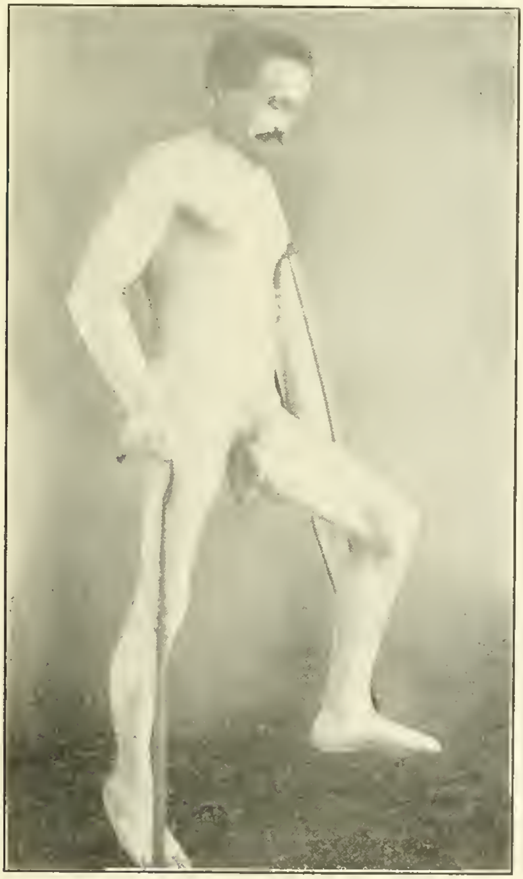

Fig. 6.—Showing flexion of the thigh on the abdomen, and of the leg on the thigh after the operation. The man is standing on one foot and the left foot is not supported.

The anesthesia is complete on the right thigh and is in incomplete on the left thigh. The areas correspond closely with those given by Seiffer as the distribution of the second and third lumbar posterior roots. There is no sharp limitation from the sensitive area, and the portion of the front part of the thighs close to Poupart's ligament in the distribution of the first lumbar roots is sensitive. The case shows that the cutting of two adjoining roots may cause persistent loss of sensation in the distribution of these roots, or at least loss for several months.

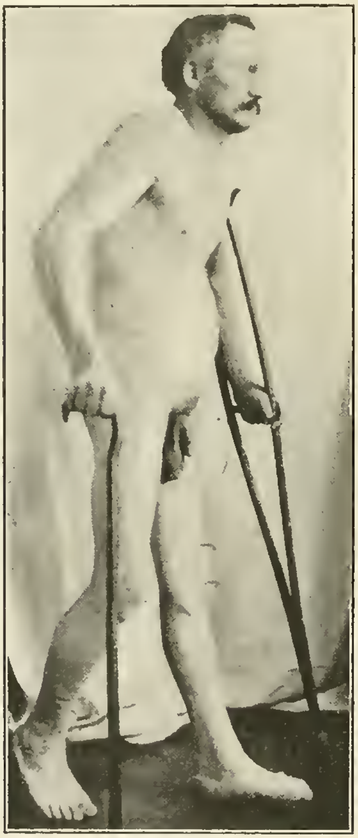

Fig. 7.—Showing that the whole foot is placed firmly on the ground after the operation. The limbs are no longer firmly adducted in walking.

The return of the patellar reflex on the left side, even though it is not in normal intensity, shows that the fourth lumbar root, if intact, may be sufficient for this reflex, although on the right side the patellar reflex is very weak. This reflex probably depends on the integrity of the third, fourth, and fifth lumbar roots, and possibly of the second lumbar root also. In this case only one root of this group, the fourth lumbar, on each side remains intact.

The area of the fifth lumbar root is normally sensitive, and this fact demonstrates what is well known, viz., that destruction of one root does not cause much impairment of sensation.

Remarks by Dr. Frazier.

Because of an existing bronchitis it was deemed advisable to perform the operation under spinal anesthesia rather than ether. Accordingly, 1.75 c.c. of a 4 per cent. solution of stovaine were injected into the spinal canal between the second and third lumbar vertebræ. Anesthesia was complete within ten minutes, and the operation was begun with the patient on his right side, head slightly elevated. The operation was under way but very few moments when the following phenomena were observed and attributed to the stovaine; the patient became cyanosed, breathing intensely labored, exclusively diaphragmatic, the pulse not rapid, but feeble; the area of anesthesia had extended up to the distribution of the trigeminal nerve. The patient felt nauseated and complained bitterly of respiratory distress. The operation was stopped, artificial respiration begun, and oxygen inhalations given, together with a hypodermic injection of atropine and caffeine. Within twenty minutes of the onset of these somewhat alarming symptoms the patient's condition improved sufficiently to warrant the continuation of the operation.

A curved incision had been made from just above the first to just below the fifth lumbar vertebra. The cutaneous flap, with its convexity toward the right, was reflected and a vertical incision in the aponeurosis made along the tips of the spinous processes. The muscles were separated on either side from those and the laminæ; the spinous processes were removed with bone forceps and the laminæ with the rongeur. The dura was incised and the first sacral nerve identified by its point of exit through the dura (on a level with the spinous process of the fifth lumbar vertebra). With this nerve identified, the sensory roots of the fifth, third, and second lumbar nerves on either side were easily found and resected. The dural wound was closed with a continuous catgut suture, and in turn the muscular, aponeurotic, and cutaneous wounds. At the conclusion of the operation

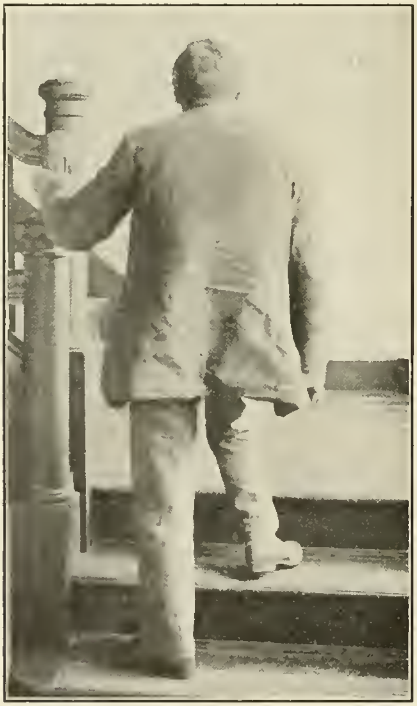

FIG. 8.—Showing the man's ability to go up stairs alone.

the anesthesia was still absolute and the patient's condition perfectly satisfactory.

The surgical aspects of the operation for division of the posterior roots of the spinal cord are virtually the same, at least in the preliminary stages, as for a laminectomy for other purposes. It is, of course, important to take every precaution to avoid contamination of the wound from the excretions and to secure primary healing. The possibility of decubitus developing after the operation and of secondary infection is quite real and not imaginary; in fact, one of Förster's cases died from this complication. We must be scrupulously particular as to the disinfection of the skin before the operation, and equally so to avoid infection afterward.

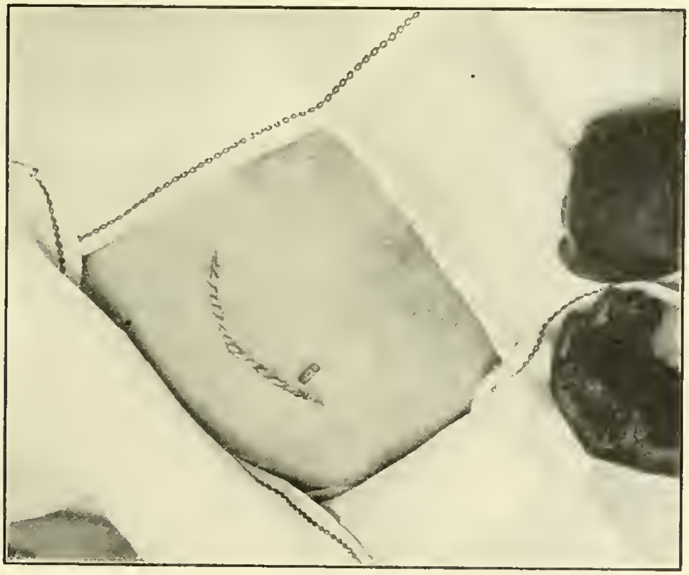

Fig. 9.—Illustration from photograph showing the direction of the cutaneous incision and counteropening for drainage.

It is necessary in the majority of laminectomies to provide an outlet for the escape of blood and cerebrospinal fluid. There is always a certain amount of oozing, and for this an outlet must be provided, but in order to prevent the establishment of a sinus communicating directly from the cutaneous surface to the depths of the wound the same precautionary measures should be adopted as we are in the habit of employing in operations for pretentorial, but especially for subtentorial lesions. The incisions in the skin, aponeurosis, and muscles should be so made that one does not lie directly over the other, and the outlet for the drainage material is provided by a stab puncture to one or the other side of the incision. These seem more or less trivial, perhaps, to the casual observer, but as a matter of fact they are the most important features of the technique of laminectomies. The cutaneous incision which I prefer is a curved, semilunar incision, the extremities of which are a little to one or the other side of the median line (Fig. 9). This includes all the structures down to the deep aponeurosis. Upon reflecting this flap another incision is made a little to one side of the spinous process from the first to the fifth lumbar vertebra. The spinal process and the laminæ are then removed and the dura opened and the roots divided in a way about to be described.

When the operation is completed the dural incision is closed with a continuous catgut suture, the erector spinæ muscles with interrupted and the aponeurosis with chromic catgut sutures; the skin incision with a continuous silk and horse hair suture (Fig. 10). A stab wound is then made in the skin a little to one side of the incision and a cigarette drain introduced into the depth of the wound. To insure protection of the wound the skin is painted with Whitehead's varnish. An adequate protective dressing is applied and secured in place with strips of adhesive plaster.

There are a few other technical points that merit comment; the selection of an anesthetic is a matter of some importance, especially as in my case, when the patient had a severe bronchitis, when spinal anesthesia was selected. The operation was conducted under stovaine anesthesia, and it may be of interest to note that shortly after its injection the patient's condition caused us not a little apprehension. Although a moderate dose was used and the injection made between the second and third lumbar vertebræ, within a few minutes the area of anesthesia extended up to the distribution of the trigeminal, the pulse became weak though not rapid, and the breathing so seriously embarassed that artificial respiration was resorted to. All the muscles of respiration except the diaphragm were paralyzed, and the action of the latter hardly sufficient to prevent asphyxiation. Artificial respiration, oxygen inhalations, and hypodermic injections were continued for fifteen or twenty minutes when the patient had revived sufficiently to warrant the continuance of the operation. It may be interesting to note in passing that the accessory respiratory muscles did not resume their function altogether until at least six or seven days after the operation.

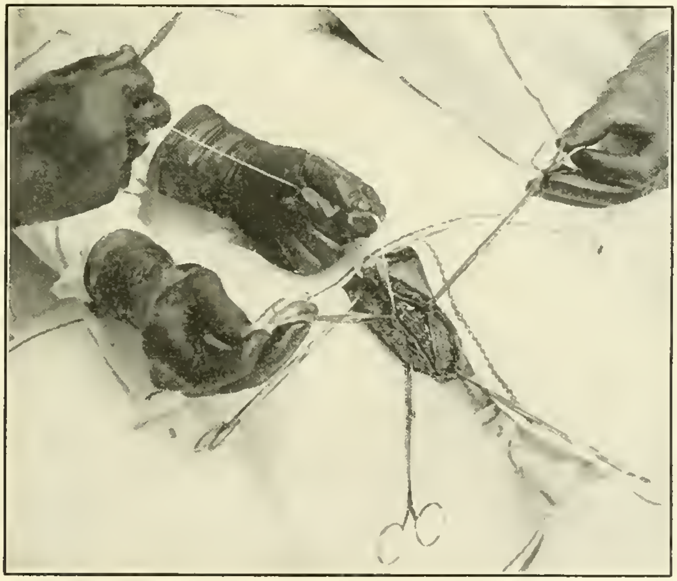

Fig. 10.—Illustration from photograph showing the muscle layer already closed with interrupted sutures, a continuous suture being introduced into the aponeurosis.

The position of the patient is not of minor consideration. There have been instances where the patient died in collapse on the table, as did one of Tietze's cases, and this catastrophe must be due, in part at least, to the loss of so much cerebrospinal fluid. As a precautionary measure, therefore, especially when the seat of operation is the lumbar region, the head of the table should be lowered so that the hips and pelvis are well above the head and neck. This reduces to a minimum the escape of cerebrospinal fluid, which in the course of a long operation would be not inconsiderable, and at the same time minimizes that more or less troublesome venous hemorrhage from the plexus of dural veins.

There has never seemed to me any valid reason for dividing the operation into two stages unless the condition of the patient at the time would seem to demand it, an experience I have never had, and yet in the European clinics this seems to be a very common practice in operations both on the head and spine. In practically all the cases reported by Förster and Tietze, Gottstein and Kuttner, this method was adopted. It is claimed for the two-stage operation that there is less bleeding during the second stage and therefore a more satisfactory exposure of the structures. As a matter of fact, hemorrhage is easily controlled by a small tampon or strip of gauze laid along either side of the dural incision. At either extremity of the edge of the dural flap on either side a silk suture is introduced; by traction on these the dural flaps are held firmly against the tampon (Fig. 11). Should there be any leakage of blood into the canal during the operation it should be removed by irrigation and not by sponging, in order to avoid every possible source of trauma, and before the dural wound is finally closed every vestige of blood should be removed in the same way, as a small clot, if allowed to remain, might cause undesirable pressure symptoms. It is needless to say that once the meninges have been opened one must be scrupulously careful to avoid traumatizing the cord and its roots. The avoidance of trauma, of hemorrhage, and infection are absolutely essential to success.

As to what roots should be divided, this will depend upon the individual ease; in our own it was the second, third, and fifth lumbar roots. One must have a way of distinguishing one root from another, or at least of being able to identify any one of the several roots exposed, and Förster's plan is simple and practical. Before the spinous processes are removed a pin or suture is introduced into the skin to one side of the incision on a level with the spinous process of the fifth lumbar vertebra. This corresponds to the level at which the first sacral nerve leaves the dural sac. With any one nerve identified it is a very simple matter to identify the others.

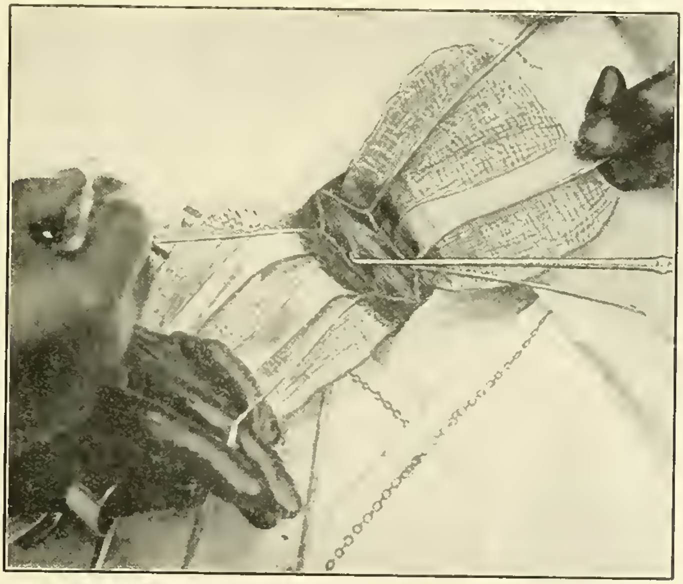

FIG. 11.—Illustaation from photograph showing the arrangement of gauze pads, traction sutures, and a hook passed around both the anterior and posterior roots.

The actual division of the sensory roots is not a difficult though a delicate procedure. My apprenticeship was served many years ago when for experimental purposes I divided a number of posterior spinal roots on dogs. Here the roots were much smaller and not so easily separated. To distinguish the posterior from the anterior roots one must remember that the posterior root has its origin from the posterior part of the cord, and is divided into several bundles. The two roots are elevated on a blunt hook and drawn gently toward the median line; when the arachnoid is divided the slit between the two roots is not difficult of recognition. The posterior root is then isolated on a second smaller blunt hook, the first hook withdrawn, and the posterior root either divided or resected. As the sensory root at its entrance into the cord loses its neurilemma sheath, it will not regenerate when once divided, so that resection is unnecessary.

As to the condition of the patient after the operation, it is of interest to note that the function of the respiratory muscles, which was inhibited by the stovaine, was not fully restored until the sixth or seventh day after the operation. This must be a very unusual sequela of spinal anesthesia. Since this experience I had an opportunity to observe, following a high injection (between first and second dorsal vertebræ) of stovaine for an amputation of the breast, somewhat similar phenomena, but they lasted only a few moments. That the muscular inhibition should have persisted for a week is difficult to account for. For a week or ten days the patient complained of paroxysms of sharp pain and twitching in the extremities, but these were attributed to the necessary manipulations of the spinal roots. They were, however, a very conspicuous feature and caused, for the time, considerable discomfiture.

The after treatment of these cases differs in no respect from that of laminectomies for other purposes. The patient should remain in bed two weeks, but at the end of the first week the patient is allowed to sit up in bed, at least if the wound is closed and there is no further escape of cerebrospinal fluid. For two or three months a light brace should be worn.

Extracted from the American Journal of the Medical Sciences, June, 1910

THE TREATMENT OF SPASTICITY AND ATHETOSIS BY RESECTION OF THE POSTERIOR SPINAL ROOTS.[1]

By William G. Spiller, M.D.,

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Cases of spasticity are very numerous, and in some instances the spasticity is far greater than the motor palsy; indeed, if the implication of the pyramidal tract be slight, the spasticity, in the opinion of some, is greater than when the degeneration is intense. Spasticity is affected by various conditions. Sensory irritation, such as cold, faradic electricity, handling of the body, or voluntary movement increases the spasticity; and sleep may diminish it; so that spasticity may be less in the morning than in the evening. These effects are produced through the peripheral nerves.

Irritation of peripheral sensory fibers, as in joint disease, may increase the tonus and cause contracture. It is common to find the tendon reflexes exaggerated in the wasted limb in arthritic muscular atrophy.

Athetosis is essentially a form of spasticity, differing from ordinary spasticity chiefly in the varying degree of tonicity in the different muscles. An athetoid limb is one necessarily spastic, and first one group of muscles, then another, exhibits a predominance of the spasticity. This view has been shared recently by Schwab and Allison;[2] they state that athetosis and spastic conditions of organic origin are essentially similar processes; that athetoid movements may be regarded as variants of permanent tonic spasticities.

In order that we may have clearly in mind the rationale of the method of treating spasticity by resection of the posterior roots, it may be well to give at this juncture an elementary sketch of the chief factors concerned in spasticity.

Muscular tonus depends on the integrity of the peripheral sensory nerve fibers, of the posterior roots, of the cells of the anterior horns, and of the peripheral motor nerve fibers. Fibers pass through the brain and cord and exert a controlling influence over the spinal reflexes. These fibers have an inhibitory effect, and act as a damper on the spinal reflexes. In contrast with these are possibly other fibers whose function is to accelerate the tonus, "bahnende" fibers of the German writers. It may be partly from irritation of the latter that in slight lesions of the pyramidal tracts tonicity may be greater than in complete destruction of these tracts.

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[1] A lecture delivered at the University of Pennsylvania during "Home Coming" week, March 29, 1910.

[2] Journal of Nervous and Mental Disease, August, 1909; Journal of the American Medical Association, February 12, 1910, p. 551.

The inhibitory effect of the cerebrum on the spinal reflexes is probably accomplished through the pyramidal tract. Afferent impulses are passing constantly from all parts of the body below the head to the spinal cord, and exert an influence on the cells of the anterior horns of the spinal cord, by means of which motor impulses are sent out to the muscles. It is, indeed, fortunate for us that many of these reflex acts do not enter our consciousness. When standing in a rapidly moving car we may be engaged in reading, and yet are constantly and unconsciously altering the tonicity of opposing groups of muscles. A wooden figure of the same proportions as the human body placed in a similar position would soon fall over. During life our muscles are constantly in a state of tonicity suitable for the various functions they are called upon to discharge, but in disease this condition of well-adjusted tonicity may be altered. A soldier standing "at attention," as Strümpell has said, exhibits the most perfect movement.

When some lesion damages the pyramidal tract the inhibition of the brain is removed and the spinal reflexes are left without control. The desideratum would be the restoration of the pyramidal tract, but that manifestly is impossible. The next best thing is to remove some of the afferent peripheral impulses, so that the cells of the anterior horns may not be kept in a state of overexcitement. It is possible that in lesions of the pyramidal tract the excessive tonicity is caused by the cerebellum, and theoretically it might be desirable to operate so as to influence the action of the cerebellar nuclei. An operation may be devised some day with this end in view, although at present it does not seem possible, and yet in 1905 the proposal of division of the posterior roots to influence spasticity met no response.

In this connection I quote from J. S. Risien Russell's Lettsomian Lectures:[3] "As sensation does not accrue as a result of all peripheral stimuli which evoke a central (reflex) action, Sherrington adopts 'receptor' as the neutral term to apply to the peripheral apparatus, which receives the stimulation. He points out that there are two main fields of distribution of the receptor organs—a *surface field*, which is situated at the surface layer of the organism, but external and internal, which he speaks of as exteroceptive and interoceptive respectively; and a *deep field*, which is situated in the tissues of the organism beneath the surface layer, which he designates proprioceptive. The stimulations which occur in the deep field are traceable to actions of the organism itself, as opposed to stimuli received from without. Hence the term 'proprioceptor,' which he has applied to these stimuli, which are brought about largely, as he says, through the agency of mass, with its mechanical consequences of weight and inertia, and also largely through mechanical strains and alterations of pressure, resulting from contractions and relaxations of muscles. Among the functions subserved by the receptors of the proprioceptive, or deep field, is that of maintaining a state of tonic reaction in the skeletal muscles, which Sherrington regards as concerned with the maintenance of attitude. . . .

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[3] British Medical Journal, February 19, 1910, p. 425.

"Bouché has determined that fits induced by intravenous injection of absinthe in an animal deprived of one cerebral hemisphere were clonic and mainly flexor in type from the intact cerebral hemisphere, whereas the fit on the other side was tonic and chiefly extensor. Section of the mesencephalon during the fits abolished the clonic discharges, and only allowed the tonic to continue. Even more striking are the results of Leonard Hill, who was able to alter the character of the fit from tonic to clonic at will, according as he interrupted the arterial supply to the cerebrum by clamping the vessels or allowed the blood charged with absinthe to reach the brain. . . .

"Sherrington has shown that removal of the cerebrum results in what he speaks of as decerebrate rigidity, which fully accords with Dr. Jackson's view of cerebellar influx. Moreover, he found that this rigidity could be abolished, and was replaced by flaccidity in the homolateral limbs on hemisection of the spinal cord in the cervical region, which makes it evident that tonus is derived from the mesencephalon or cerebellum. Sir Victor Horsley, acting on Dr. Jackson's suggestion, divided the mesencephalon at the level of the intercollicular groove, and thus allowed of decerebrate rigidity appearing. Removal of successive sections of the cerebellar cortex produced no appreciable effect on the hypertonus until the sections involved the intrinsic and paracerebellar nuclei [nuclei of Deiters and von Bechterew]. A large horizontal lesion, separating the dorsal half of the cerebellar cortex without involving the nuclei, was also made three weeks before the division of the mesencephalon, again without any appreciable effect on the decerebrate rigidity which resulted in these animals, as compared with that which obtained when a similar section of the midbrain was performed in normal animals. Thiele, in his researches undertaken with a view to determine the source from which the hypertonus which constitutes Sherrington's decerebrate rigidity is derived, found that the chief source of this energy was at the level of the paracerebellar nuclei. Successive lesions of the mesencephalon, commencing in front and proceeding caudalward, failed to affect the decerebrate rigidity until the nuclei were reached. The experimental evidence is thus convincing, both as proving that the nuclear region of the cerebellar complex is the source of the energy, which constitutes the cerebellar influx, and as thus confirming the view enunciated by Dr. Jackson more than thirty years ago. . . .

"All the facts that have been established in regard to the cerebellum are in accord with a view that its cortex is concerned with the reception of afferent impressions which are derived from deeper structures like the muscles and joints, as well as those which the organ receives from the labyrinth, and that efferent impulses are thus evoked from its muscles, which are responsible for that degree of tonus in the general musculature of the organism which is essential for the maintenance of attitude, and which makes it possible for coördinated movements to be executed."

These important statements show that tonicity of muscles depends on the cerebellum, and probably on its nuclei, and less so on the cerebrum. The connections of peripheral afferent fibers with the cerebellum is by means of the posterior roots. It is probable that in excessive tonicity destruction of the cerebellar nuclei would produce flaccidity. By cutting the posterior roots from a spastic limb we weaken or destroy this spasticity by removing the impulses to the cerebellum, on which organ it depends, and therefore the operation has a logical basis. Were we in a position to increase the cerebral inhibition over the motor tracts, we should not resort to resection of the posterior roots.

Various attempts have been made to overcome spasticity. In a paper read in 1905 with Dr. Frazier[4] before the New York Neurological Society on the treatment of cerebral palsies and athetosis, nerve anastomosis was discussed. At that time I recommended the resection of the posterior roots for the relief of spasticity and athetosis, but the idea was so novel it called forth no discussion, and receiving no encouragement, I hesitated to advise further an operation that seemed too much like an experiment on man, and one not without danger. We therefore contented ourselves with an attempt to relieve spasticity and athetosis by anastomosis of one peripheral nerve with another, hoping that we might satisfactorily diminish afferent impulses, although we knew that we must diminish efferent impulses, but felt that sufficient efferent motor impulses would be left for fair discharge of function. The results seemed at first very promising. The most extreme spasticity and athetosis yielded to this treatment, but the young man on whom the operation was performed deserted the hospital and withdrew himself from our observation. When we saw him again, some three years later, athetosis in the operated limbs had almost disappeared, but contracture had become pronounced and interfered greatly with voluntary motion.

An attempt similar to ours has been made by Nutt[5] recently.

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[4] Journal of Nervous and Mental Disease, May, 1905; American Journal of the Medica Sciences, March 1906; University of Pennsylvania Medical Bulletin, January, 1910, p 314.

[5] American Journal of Orthopedic Surgery, November, 1909, p. 151.

This author believes that much may be gained by cerebral rest. His method differs from ours only in uniting the cut ends of the same nerve instead of uniting the cut ends of different nerves.

Schwab and Allison have proposed another method of treatment for the relief of spasticity. Their method is described as follows: "We have been led to devise a method which we shall refer to as muscle group isolation. This implies the isolation of the muscle or the group of muscles which are at fault in the production of contracture, deformity, or athetosis. It is made effective by cutting off from the central nervous system the connection along which the abnormal impulses, active in causing spasticity or athetosis, are transmitted. This is done by a direct attack on the nerve itself, by isolating it, and injecting it with an alcoholic solution. There has resulted in the cases to be described an immediate paralysis of the physiologically stronger group of muscles without interfering with the free muscular use of the antagonists. At this point physiological exercises planned to further strengthen the antagonist may be used."

Schwab and Allison have recently (February 12, 1910) stated that they regard their method as holding considerable promise of relief. They have not attempted to cure spasticity by alcoholic injections into the peripheral nerves. They believe that by their method the peripheral pathway of the spastic impulses being temporarily blocked, their return in the original sum might be lessened.

More promising than any other method for the relief of spasticity has seemed to me for some five years the resection of posterior roots innervating the spastic limbs. He who studies disease probably has many indications justifying this operation. So far back as 1893 or 1894 I observed return of the patellar reflex in tabes after hemiplegia had occurred. This return of reflex irritability indicated that the degeneration of the posterior lumbar roots in this case of tabes had been sufficient to abolish the reflex but that enough fibers were retained in the posterior roots to permit a return of this reflex when cerebral inhibition over the spinal reflexes was removed by degeneration of the pyramidal tract. The opposite conditions to those described in this case have been observed repeatedly. When degeneration of the pyramidal tracts has been followed by degeneration of the posterior columns extending well into the lumbar region, in posterolateral sclerosis, spasticity and exaggeration of tendon reflexes have given place to flaccidity and diminution of tendon reflexes.

Unquestionably the first operation for the cure of spasticity by division of the posterior roots was made by O. Foerster's directions.[6] His first paper was published in 1908. He relied on Sherrington's experiments on monkeys, by which it was shown that resection of one or two posterior roots produced no anesthesia, and that the sensation of any cutaneous area depends on three roots, and that not until three adjoining roots are cut does anesthesia occur. As yet satisfactory investigations concerning man are wanting, although it has been demonstrated that division of a single posterior root causes no sensory disturbance. Bruns believes that more than three roots may supply any cutaneous area. Foerster has never had more than two adjoining roots divided.

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[6] Zeitschrift für Orthopädische Chirurgie, 1908, xxii, 1 to 3; Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie, 1909, xx, 493.

For the report that he makes in his second paper he selected two cases of congenital spastic diplegia, one case of tuberculous cervical spondylitis, one case of multiple sclerosis, and one case of hemiplegia. After the operation a certain resistance of the muscles persisted for several weeks in passive movement and extension of the limbs. This resistance he attributes to the pain from the irritation of the posterior roots produced by the cutting. He found at first after the operation that the leg was flexed at the knee and the thigh flexed on the hip, rotated inward, and adducted. This position alters as the cut roots degenerate, as does also the resistance of the muscles in passive movement. In spastic paralysis cutting of tendons or muscles seldom is successful. In some cases tenotomy may be necessary after division of the roots. Foerster recommends that the limbs should be placed in some corrective apparatus after division of the roots, to avoid faulty position.

The Achilles reflex was lost after the operation in Foerster's cases. He attributes this to the cutting of the second sacral and possibly also of the fifth lumbar root. It is not known positively in which posterior roots the Achilles reflex arc lies, but Foerster places it from his cases in the first and second sacral and possibly fifth lumbar roots.

Tietze[7] says the operation is not very dangerous. He had five cases with one death, and death in this case was by infection from decubitus. Gottstein had two operations and Küttner five operations and no deaths. It is, however, recognized as a major operation. Tietze refers to another fatal case in which collapse occurred before the roots were cut.

Successful cases treated by this means have been reported by Clarke and Taylor,[8] and Taylor has diminished the seriousness of the operation by devising unilateral laminectomy.

Dr. Frazier and I have reported one case in detail in which the results of the surgical treatment have been most satisfactory. Since the publication of that report the improvement in the patient's gait has been marked. He has been receiving systematic training in walking from Dr. Wm. Burdick.

The patient referred to at a previous part of this paper, on whom nerve anastomosis was performed, has also been treated by this method of resection of the posterior roots by Dr. Frazier, and in this case the cervical roots were cut. Improvement in this case also is distinct, but tenotomy may be needed on account of the shortening of the flexor muscles of the elbows.

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[7] Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie, 1909, xx, 559.

[8] New York Medical Journal, January 29, 1909, p. 215.

Resection of posterior roots is useless when paralysis is intense.

In regard to some of the objections made to the treatment, I acknowledge that the operation is serious; had it not been so I should not have remained contented in 1905 in merely proposing this operation, but because of its seriousness we hesitated to carry out the suggestion. Cortical rest produced by peripheral operations seems to me a condition of little weight in the treatment of spasticity.

The overlap of sensory distributions is considerable, but this does not mean that all the posterior roots of a limb must be cut to lessen spasticity. In the lumbar region the roots at their origin are near together; and in the cervical region, though not so near as in the lumbar region, they are still close to one another, and therefore exposure of a very large area is not necessary. No one would desire complete sensory paralysis from cutting the posterior roots; a paralysis of this kind would be unnecessary, and would probably cause incoördination and defeat the object of the operation. Indeed, because of the overlapping of root distributions loss of sensation is not likely to occur. Considerable impairment of sensation developed in our first case, but this was probably because the man had had previously great impairment of sensation from his injury, and while sensation had returned, the fibers within the cord were far from normal, and therefore after the resection of certain posterior roots too few afferent fibers were left for the maintenance of sensation. Little or no disturbance of sensation was caused in our second case by extensive resection of posterior cervical roots.

In conclusion, I would say that a means has been devised full of promise for selected cases of spasticity and athetosis, and that these conditions which formerly perplexed and baffled us have in a considerable measure yielded to treatment. Undue enthusiasm is to be deprecated, as bringing the method into disrepute. Cases must be carefully selected, and only those are available in which spasticity is great and weakness is comparatively slight. The association of athetosis need not be a deterring factor, as the strength of a limb often is increased by the excessive muscular action of athetosis. As in every new method, caution is needed, and proper valuation can be placed upon the method only when more experience has been obtained.

in the upper part and the back of the thighs. There were never any symptoms in the upper part of the body, the head, or the upper extremities.

The pain and weakness have increased to the extent that the patient is confined to his bed and can only move about with the greatest difficulty, and by aid of crutches. Paresthesias, tingling, and burning sensations are present in the left leg below the knee, and sometimes in the right leg and the left thigh. Pain in the back and legs has become worse. There was never any disturbance of the bowels or the bladder.

The heart and lungs are normal. The head and the cranial nerves are normal. There is no ankylosis of the jaw, nor trophic disturbances of the upper part of the body, the head, or the extremities. There is no wasting or deformities. The upper extremities show no sensory or motor involvement, and no ankyloses. Active and passive movements are normal. The biceps tendon reflex is slightly increased on either side. The triceps jerk is about normal on both sides. There is considerable tenderness on pressure over the muscle bundles on either side of the spine, but this is particularly marked on the right side in an area extending from the tenth dorsal to the second lumbar vertebra, and for a few inches laterally along the last ribs. The spine is not completely fixed as there is some power of rotation with slight ability to flex and extend the spine, but these movements are only in the dorsal and cervical regions; any attempt to move the lower spinal vertebræ causes intense pain.

Sensory changes: The patient, while at first only complaining of paresthesias in the lower legs, now has these sensations in the thighs and a feeling of constriction around his waist. The nerve trunks in the calves of the legs and just above the knees on both sides are very tender upon pressure, a condition having appeared only in the last few days. Tactile sensation is not impaired in any part of the legs. Pain sensation by the pin point test is absent in an area over the anterior part of the right thigh, extending down to the knee. There is also a similar area in the left leg, extending from just above the knee to about three-fourths of the distance to the ankle and half way around the front of the leg. This area is continued still further to the ankle as an area of hypalgesia. Heat is not recognized as such (by test tube method) in an area on the right lower leg extending from just above the knee downwards over the lower leg, with the exception of a small area of retained sensation on the middle posterior part of the thigh (area about 3 inches by 2 inches). Changes in the left leg are about the same as the above except that here the area begins just below the level of the knee and there is no spot of normal perception posteriorly.

Cold is recognized as cold over all portions of the legs but in the areas mapped out above the quick responses are not elicited as in the other portions of the extremities. The muscles in both thighs are weak and movement is impaired, and in attempting to move his legs the patient complains of pain in the hip joints and back and in the muscles of the thighs. Movements in the hip joints are stiff and restricted and very painful. When first admitted movements were not so restricted and the impairment was due only to muscle spasm, but at present there appears to be a slight degree of ankylosis. Both lower legs are atrophied and cold and have a doughy appearance with glossy hairless skin. The feet are somewhat cyanosed and swollen, but do not pit on pressure. The power in the right lower leg muscles is diminished, especially in the extensor and peroneal groups and there is toe drop. This can, however, be overcome as patient has some slight remaining power in the extensors. The left lower leg shows great loss of power in the extensor and peroneal groups, and weakness in the flexors, and there is permanent toe drop. There is a very slight power of movement in the extensor brevis digitorum.

Electrical Examination: In the muscles of the right thigh and lower leg the response is slow but in normal series. Examination of left thigh and leg gives a reaction of degeneration (An Cl C> Ca Cl C) in the quadriceps group, hamstrings, and the anterior tibial and peroneal extensors of toes. The flexors of the toes and the muscles of the calf and the extensor brevis digitorum give normal but slow response.

Case II.—E. O., a teamster, age 33 years, was admitted to Philadelphia General Hospital Aug. 24, 1909, complaining of pains in the back and shoulders. There was nothing of importance in the family history. One year ago the patient had what probably was a chancroid, having had no secondary symptoms of any character, and no other venereal disease. At the age of eight he had pain in his knees, this, however, was not of acute rheumatic type, and as there are some of the other signs of rachitis present the pains in the knees were probably rachitic in origin.

The present illness began about four months ago (May) when the patient upon awakening had severe pains, sharp and stabbing in character, in the mid-dorsal region. This pain was not present when the patient lay quiet, but upon making any movement, such as turning or sitting up in bed, it became very severe, as it was also at times when the patient drew a deep breath. The pain always has the same sharp, stabbing character and extends along a line just to the left of the vertebræ, sometimes involving the neck and head. It does not radiate laterally or into the extremities. The patient has lost 17 pounds in four months.

Examination: The heart is normal. The lungs show consolidation of the right upper lobe. There are bony prominences at the left sternoclavicular articulation and at the costocartilaginous junctions probably rachitic in origin. The head and cranial nerves, with exception of disturbance in sensation, which will be mentioned later, are normal. There is no ankylosis of jaw.

The upper extremities: With the exception of impairment of motion in the left forearm, due to a previously fractured radius, the arms show no impairment or restriction of active or passive motion. Within the last month, however, the left shoulder joint has become to a certain degree ankylosed and interferes with the free movement of the arm. The reflexes are normal on the right, absent on the left for the above reasons.

Lower extremities: No impairment of active-passive movements is noted. The patellar tendon reflexes are somewhat exaggerated. The Achilles jerk is normal. There is no Babinski.

Sensation: Pin prick, over entire body, is only recognized as dull. Over an area including the face, anterior part of neck, and body down to the lower costal border, also both arms and shoulders with exception of the interscapular space and a small area on upper, outer, anterior, and posterior surface of the left upper arm. The patient recognizes cold as warm. On external upper surfaces of both thighs the patient distinguished cold and warm, but not so well as in the other normal areas. The palms of the hands are also normal. Cold is called warm in the right lower leg and this area extends up to about six inches above the knee, with a further extension upwards of a couple of inches on the external (lateral) aspect of the thigh. The left lower leg has the same sensory changes but the area only extends above the knee in two narrow strips internally and externally situated, and extending to about four inches above the knee.

Vertebral column: There are stiffness and restricted movement of the vertebræ of the dorsal, the lumbar, and to a lesser degree of the cervical regions. Tenderness, upon pressure, is elicited in the muscles of the back to the left of the vertebræ and from the angle of the scapula to below the pelvic crests, and is most pronounced in the lumbar region.

Case III.—W. A., a painter, age 57 years, was admitted to Philadelphia Hospital Sept. 17, 1909. He complained of pain in the right shoulder and arm and limited motion and weakness of the right leg. The early medical and family histories contain nothing of clinical importance. He fell upon his back from a high ladder about a year ago and has had a stiff back ever since. He also fell from a trolley car in March, 1909, hurting the arm and shoulder and has at present a circumflex and musculospiral paralysis. About two months ago (July) the patient's right foot and ankle became swollen and painful and has remained so ever since. He cannot move the right leg and foot around in the bed. Two weeks ago he began to have dull pains in the hips, back, and back of the neck.

Examination: The heart and lungs are normal. The head and cranial nerves are normal. The left arm is normal in power and mobility, and the reflexes are normal. The right shows the characteristic changes of the peripheral palsies mentioned above. Upon electrical examination, besides reaction of degeneration in muscles supplied by musculospiral and circumflex nerves, there was diminished response to the faradic current in the infraspinatus and cervical part of the trapezius on the right side.

The left leg is normal. The right leg shows stiffness in the hip and knee joints with dull pain in the right hip and the bottom of the foot when patient tries to stand. Power is diminished though there are no paralyses of the muscles. The patellar tendon reflexes are exaggerated and crossed adductor contraction can be elicited. The Achilles jerk is also exaggerated, with at times a clonus. Flexion of the thigh and leg causes reflex rigidity and pain. The cremasteric, abdominal and plantar reflexes are normal.

Sensation: The temperature sense seems to be impaired except in the face and hands, as patient frequently mistakes hot for cold and *vice versa*. No tactile or pain disturbances occurs except in the right arm, where it is due to the peripheral palsy. The sternoclavicular joints are enlarged, as are also the costocartilaginous junctions of seventh and eighth ribs. The transverse processes of the cervical vertebræ, especially the sixth, are elongated and enlarged.

A few days after admission the patient developed a stiffness of the jaw. interfering with the movements in opening and shutting it. There is stiffness extending over the entire vertebral column and causing impairment of motion and stiffness in the patient's gait.

All three of these cases seem to conform to the type described by V. Bechterew, the root symptoms being the predominating and in the first case even causing a distinct muscular atrophy and double toe drop, this condition being permanent in the right leg. The possibility of palsies occurring in this condition has been mentioned by several writers, but I can find only one case in the literature, recorded by Heiligenthal,[1] in which reference was made to a peroneal palsy.

The trophic changes found in the lower extremities, in the first patient, brought to mind the possibility of the presence of a sclerodermal change; attention having been called to this possible complication or accompaniment of rheumatoid arthritis by Dercum,[2] the changes here present, however, though rather suggestive, were not sufficient to warrant such a diagnosis. That the above cases were not classical cases of the Von Bechterew's type can be seen when the involvement of the proximal joints is taken into consideration, such as was present in these cases and is characteristic of the Marie-Strümpell type. This brings up the question as to the probability of all forms, in time merging, more or less, into each other, and I do not believe a distinct type, as a rule, to exist throughout the entire course of the disease.

A fact of some importance, observed by Heiligenthal[1] and present in one of the above cases, is the intermittence or change in the condition of the ankylosed joints. This may go to such a degree that a joint apparently very stiff may later become practically free and then again become involved. This intermitting, if a patient were to be examined by different observers at different times, would no doubt lead to some confusion as to the type of the disease. Difficulty might occur in the differential diagnosis between this disease and syringomyelia, due to possibility of occurrence of dissociation of sensation together with spinal rigidity and kyphosis. This should, however, by careful study leady to no error in diagnosis as the signs, other than those mentioned, are in no ways similar.

The cervical exostoses in the third case, with the rheumatic diathesis and the rachitic enlargements, are interesting as having possibly some connection with a condition of body in which there is tendency toward this disease.

1. Heiligenthal, H.: "Recognition of Spondylosis," etc., *Deutsch Zeitsch. für Nervenheilkunde,* 1900.
2. Dercum, F. X.: "Scleroderma and Chronic Rheumatoid Arthritis," *Journal of Nervous and Mental Diseases,* Vol. XXV, No. 10.

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MENINGITIS AND DISEASE OF THE RADICULAR NERVES IN TABES DORSALIS.*

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(Plates XVIII. and XIX.)

The changes in the radicular nerves in tabes, the relation that meningitis bears to this disease, the character of the meningitis, its relation to syphilis, and the part the meningitis plays in the causation of the radicular nerve changes, are all questions that have been discussed at length in the literature without, however, having been definitely answered.

With a view to throwing some light upon this subject an opportunity was offered me, through the courtesy of Dr. William G. Spiller and others, to study the available material at the Laboratory of Neuro-Pathology of the University of Pennsylvania, which, with my own cases, consisted of the spinal meninges of seventeen cases of tabes dorsalis, the cortical meninges of sixteen of these, and the radicular nerves of eight cases of tabes and one of syphilis of the nervous system.

Twenty-five radicular nerves from eight cases were cut in serial sections and stained by hematoxylin and acid fuchsin, and by the Weigert method. Of these cases nine have already been reported, although not in this relation.

Case I. — M. M., aged sixty-six, history of syphilis thirty-five years previously. First symptom twenty-five years before death. Argyll-Robertson pupil; pains in the legs; knee jerks absent; Romberg's symptom marked.

Typical tabetic degeneration of the posterior columns, slight in degree. Slight border degeneration. Degeneration of the anterior and posterior roots. Intense round-cell infiltration of the pia. The blood vessels of the pia were thickened, the lumen narrowed, and in places intense proliferation of the inner coat of the blood vessels was present. The infiltration of the pia showed no predilection. The round-cell infiltration was typically specific in appearance. The pia of the paracentral region was thickened and infiltrated with connective-tissue cells.

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Case 2. — F. K., aged forty-eight. Family and previous histories negative. Venereal history denied. Symptoms of four years' duration consisting of lancinating pains, Romberg's symptom, failure of eye-sight, trophic ulcers of the plantar surface of the feet, absent knee jerks and other tendon reflexes of the legs; double ptosis; some slight sensory changes and Charcot's joint. The pupils responded to light and in accommodation. There was wasting of the thenar and hypothenar eminences and the dorsal interossei of both hands.

There was typical tabetic degeneration in the posterior columns, moderate in degree, and very slight in the cervical region. Both anterior and posterior roots were degenerated. There was a well-marked round-cell infiltration of the pia, and slight thickening of the vessel walls. The infiltration was not more marked anywhere excepting in the pia of the anterior longitudinal fissure. The pia of the paracentral region showed infiltration of connective-tissue cells and plasma cells. Little or no blood-vessel disease was demonstrated, although one vessel was obliterated.

Case 3 (reported by Carl D. Camp, M.D., University of Penna. Bulletin, January, 1905). — W. H., aged forty-seven. Specific infection denied. Ataxic symptoms for eight years. Ataxic station and gait; absent knee jerks; incontinence of urine; Argyll-Robertson pupil; sensory disturbances; atrophy of the muscles of the lower extremities, and wasting of the muscles of the arms.

There was advanced degeneration of the posterior columns. Very slight round-cell infiltration of the pia of the cervical and thoracic regions. The pia in the paracentral region was profoundly infiltrated with connective-tissue cells, and thickened. There was no perceptible blood-vessel disease.

Case 4 (reported by Cohen and Spiller, in American Journ. of Med. Sciences, 1901, No. 122, p. 170). — Man of sixty-seven, with a history of syphilis. The symptoms were nocturnal incontinence of urine; ptosis of the right eyelid; difficulty in mastication and deglutition; atrophy of the tongue; sensory disturbances; lancinating pains; inequality of the pupils, and loss of reaction to light and accommodation. Atrophy of the upper limbs. No ataxia of the legs. Knee jerks preserved. A diagnosis was made of cervical and bulbar tabes.

There was degeneration of the posterior roots of the spinal cord in the middle and upper thoracic regions on the right side, and in the lower cervical region, but hardly perceptible in the lumbar and lower thoracic regions. There was very slight degeneration of the columns of Burdock in the upper thoracic and cervical regions. Atrophy of the inferior part of the left twelfth nucleus was present. Intense degeneration of the left descending root of the ninth and tenth nerves. Degeneration of the seventh and sixth, the spinal root of the fifth, and atrophy of the nuclei of the oculomotor nerves.

Examination of the meninges from the thoracic and lumbar regions made by myself in this case showed round-cell infiltration of the pia, lymphoid and connective-tissue in type, mainly the latter. This seemed to be general and showed no predilection as to position. There was some perivascular round-cell infiltration of mononuclear type. The blood vessels were not thickened. The pia from the paracentral region was slightly thickened and infiltrated with connective-tissue cells. No vascular disease was observed.

Case 5 (reported by C. D. Camp, M.D., University of Penna. Med. Bulletin, January, 1905). — J. A., aged fifty-five. Symptoms of six years' duration. Romberg's symptom; Argyll-Robertson pupil; ataxia of the arms and legs; no atrophy; absence of knee jerks; lancinating pains and sensory disturbances. Besides intense lesions of tabes there was degeneration in the posterior part of each lateral column, Camp thought, of vascular origin.

My own examination of these specimens showed, besides a slight border degeneration, round-cell infiltration of the pia, which, however, was so slight as not to be pathological. The pia of the paracentral region was slightly thickened, and markedly infiltrated with connective-tissue cells and some polynuclear lymphocytes. The blood vessels were thickened as were also the walls of the blood vessels in the pia of the cord.

Case 6 (case of Dr. C. H. Potts). — M., aged fifty. Syphilitic history. Ataxia of eight years' duration; Romberg's sign; absent knee jerks; Argyll-Robertson pupil; gastric crises.

Intense tabetic degeneration of the posterior columns. Slight degeneration of the anterior roots, and marked degeneration of the posterior roots. The pia was thickened and intensely infiltrated with round cells, slightly more marked around the anterior and posterior portions of cord. Perivascular infiltration was present. The blood-vessel walls were thickened, the lumen narrowed, and obliterative endarteritis was observed. The pia of the paracentral region was much thickened, and markedly infiltrated with connective-tissue cells. The blood-vessel walls were thickened.

Case 7 (reported by S. Leopold, M.D., Jour. Nerv. and Ment Disease, 1909). — G. W., aged fifty-four. History of syphilis not obtained. The symptoms consisted in the main of ataxia, lancinating pains, loss of the Achilles-tendon reflex; vesicle and rectal symptoms; myosis; preserved patellar reflexes; ataxia in both legs.

Degeneration of the posterior root zone was pronounced through all the sacral segments, and extended as high as the fourth lumbar segment. In the middle thoracic and lower cervical segments only degeneration of the columns of Goll was observed. The degeneration was more pronounced on the right side, involving the posterior root zone from the fourth lumbar segment down to the eighth.

Dr. Leopold kindly permitted me to study his sections. The pia of the cord in the cervical region was thickened and the seat of a slight round-cell infiltration, principally perivascular and showing no predilection as to position. In the lumbar region the pia was intensely infiltrated on one side, where the degeneration in the posterior columns was most intense. The blood vessels showed slight round-cell infiltration of the inner walls. The posterior roots were degenerated, the anterior roots intact. In the sacral region the pia was thickened, but there was only slight round-cell infiltration. The cells consisted in part of mononuclear lymphoid round cells, but mainly of connective-tissue cells.

Case 8 (reported by Dr. Paul H. Bicklé, Medical Record, May 8, 1909) — J. P., aged thirty-nine. Family history negative. He had been excessive in the use of alcohol, and gave a history of syphilis. The symptoms were of seven years' duration, and consisted of lancinating pains in the limbs, ataxic gait, sphincter disorders, irregular pupils, the right reacting feebly to light, the left not at all, both reacting to convergence and in accommodation; tendon reflexes absent, inability to stand or walk, and sensory disturbances.

Advanced typical tabetic degeneration in the posterior columns extending the entire length of the cord, was present. The anterior roots were preserved in the cervical, thoracic, and lumbar regions. The posterior roots, however, were markedly degenerated. The pia in the cord was thickened and infiltrated with mononuclear cells, mainly of connective-tissue type. The round-cell infiltration was moderate in extent, but marked in degree. The blood vessels were thickened in places, and the intima in some was proliferated.

There was no predilection as to position observed in the meningeal infiltration, except around the posterior roots where the blood vessels were more numerous. The pia from the paracentral region was thickened and fibrous, and moderately infiltrated with connective-tissue cells.

Case 9 (reported by John H. W. Rhein, M.D., Journ. of American Med. Assoc., February, 1905). — Male, aged fifty-seven. Family and previous histories negative except for a history of syphilis. The symptoms were Romberg's symptom; absent knee jerks; sensory phenomena; incontinence of urine and Argyll-Robertson pupils. There was also present a tremor resembling that of paralysis agitans.

Pathologically typical tabetic degeneration of the posterior columns was demonstrated. The posterior roots were degenerated at all levels. The anterior roots were intact. The pia of the spinal cord was thickened, and the seat of moderate round-cell infiltration, more marked in the mid-thoracic region than in the thoracic and lumbar regions. The cells were of the connective-tissue type, and showed no predilection as to position. The cells were also of mononuclear type, with deeply-staining nuclei resembling mononuclear leucocytes. The infiltration resembled that of syphilis. In the lumbar region the blood vessels were thickened, and in some places the lumen was almost obliterated. The pia from the occipital and frontal regions was markedly infiltrated with cells of connective-tissue type, and mononuclear cells with deeply-staining nuclei, probably specific in type.

Case 10. — J. B., aged sixty-seven. Family and previous histories negative. Syphilis denied. The symptoms began four years previously, with sudden attack of paralysis of the left side of the face. On examination he was markedly ataxic; Romberg's symptom was present; the pupils were contracted, reacting to light and accommodation; sensation was disturbed and the tendon reflexes were absent. The left nasolabial fold was deeper than the right, and the left eyelid drooped. He was generally wasted, especially in the forearms.

The pia of the spinal cord was infiltrated with cells, the nuclei of which stained deeply. The blood vessels showed proliferation of the intima, and the lumen was narrowed. The round-cell infiltration generally showed no predilection for position, although in the sacral region it was slightly more pronounced anteriorly and posteriorly. The degeneration of the posterior columns was very pronounced throughout the entire cord.

One radicular nerve, including the ganglion, was studied. Round-cell infiltration of the endo- and perineurium was demonstrated throughout the entire radicular nerve, and extending as far as the ganglion. There was only slight degeneration observed by the Weigert method. The blood vessels of the radicular nerves showed round-cell infiltration of the walls. This was also present in the meninges, but was more marked in the radicular nerves. The pia of the paracentral region was thickened and infiltrated with large connective-tissue cells.

Case 11. — A. M., aged fifty-two. First symptoms began twenty-three years before death. Upon examination there were Argyll-Robertson pupil, absent knee jerks, marked ataxia, Romberg's symptom and sensory phenomena. There were also marked spasticity of the arms and legs, a coarse tremor of both arms and legs, some wasting, and gastric crises.

Study of the spinal cord showed advanced tabetic degeneration throughout. There was marked round-cell infiltration in the meninges and anterior and posterior roots. The blood vessels were very much thickened and there was perivascular round-cell infiltration. Gowers' tracts were degenerated in the cervical region, and also the direct cerebellar tracts, and border degeneration was present. In the thoracic region the posterior degeneration was intense, and the remainder of the white matter was diffusely involved, there being marked border degeneration, but no tendency to any systemic involvement. The degeneration of the white matter outside the posterior columns was evidently secondary to specific foci. In the posterior columns it was typically tabetic. The pia of the paracentral region showed marked thickening, and was infiltrated with connective-tissue type of cells, and with mononuclear, and occasionally polynuclear, cells. The blood vessels were thickened.

Four radicular nerves, including the ganglia, were studied. In one of these there was degeneration of the fibers at the ganglion which became more marked centralward, with marked round-cell infiltration of the perineurium, together with epi- and endoneuritis, which grew more intense centralward. In another nerve studied the fibers stained well at the ganglion, where no infiltration was observed, though the perineurium was thickened. Centralward there was round-cell infiltration in the epineurium and endoneurium, and the degeneration was marked by the Weigert method. In the third radicular nerve studied no round-cell infiltration at the ganglion was seen. Slightly centralward there was moderate degeneration of the posterior roots, but no round-cell infiltration. More centralward there was marked round-cell infiltration of the endo- and epineurium. Hemorrhagic softening of the posterior roots and intense degeneration (Weigert method) was observed, the anterior roots being intact. There was no perineuritis at this level. In the fourth radicular nerve the anterior and posterior roots coming from the ganglion were intact. The perineurium was thickened but there was no round-cell infiltration. More centralward the epineurium was implicated, but the nerves showed no round-cell infiltration. The anterior roots of three of these radicular nerves were intact. This was a case of tabes complicated by cerebro-spinal syphilis.

Case 12. — M. B., aged fifty-three. Family and previous histories negative. Symptoms began three years previously with weakness in the left leg, but this gave her very little trouble until a year later when she fell and struck her knee on the curbstone. Since then it has been swollen, but not painful, though after wearing a stocking the swelling improved, but at the same time the left foot became swollen and painful.

On examination the patient showed absent reflexes; Argyll-Robertson pupil, the left pupil slightly larger than the right; some sensory disturbances; slight diminution in power in the left leg, but otherwise good power in all the muscles. (Further the notes do not go.) The symptoms were over three years in duration.

The cord presented the typical appearance of tabetic degeneration. In the cervical region the degeneration was very slight and diffuse, was only moderate in the lumbar region, and less marked in the thoracic region. The pia in the cervical region showed considerable round-cell infiltration, though not excessive. The blood vessels were somewhat thickened. The infiltration showed no preference as to situation. Border degeneration was present in the thoracic region. The infiltration consisted of large round mononuclear and connective-tissue cells. The process was more marked at the entrance of the roots where the blood vessels were intensely involved. The arteries were thickened and there was perivascular round-cell infiltration and some round-cell infiltration of the outer walls of the veins, though this process was not marked. Examination of the pia in the paracentral region showed a slight round-cell infiltration of connective-tissue type. The pia was thickened, but no blood-vessel disease was demonstrated.

Five radicular nerves were studied, in three of which the ganglia were included. In the first the posterior roots were markedly degenerated, the anterior roots only slightly so. No round-cell infiltration was observed. More centralward there was an area of softening in the posterior roots, evidently hemorrhagic in character, much blood pigment being present. The posterior roots were intensely degenerated throughout the entire radicular nerve, while the anterior roots showed no degeneration to speak of. In a second nerve the fibers stained well at the ganglia, and there was slight round-cell infiltration of the epi- and endoneurium, and slight degeneration more centralward by the Weigert method.

In a third nerve near the ganglion there was no round-cell infiltration, nor degeneration of the roots. A little more centralward there was intense round-cell infiltration of the epineurium, near which could be seen destruction of the fibers and accumulation of round cells. By the Weigert method there was observed slight border degeneration of the posterior roots, the anterior roots being intact. Still further centralward there was slight degeneration of one portion of the posterior roots, the anterior roots being intact. There was intense round-cell infiltration of the epi- and endoneurium around the posterior roots. Further centralward the anterior and posterior roots showed no degeneration whatever.

In still another nerve there was degeneration of the nerves and round-cell infiltration involving the endo- and epineurium. In a fifth nerve degeneration of the fibers could be seen coming from the ganglia in the posterior roots. The anterior roots were not degenerated. More centralward there was intense degeneration of the posterior roots, but only a slight round-cell infiltration of the perineurium. There was no epi- or endoneuritis. This did not show the typical Nageotte degeneration. The round-cell infiltration was of the connective-tissue type. The blood vessels were diseased in the radicular nerve, showing narrowed lumen. Infiltration was less marked in the radicular nerve than in the meninges.

Case 13. — T. O'M., aged forty-three. Family and previous histories negative. Patient was admitted with symptoms of advanced tabes, the first symptoms beginning over seven years ago. He was unable to walk for a year before his death. Characteristic lancinating pains were present, also ataxic gait; Romberg's symptom; absent knee jerks; unequal pupils reacting sluggishly to light and readily to accommodation, and later Argyll-Robertson pupil. The patient became unruly toward the last of his life, and finally died of tuberculosis of the lungs at the age of forty-three.

The spinal cord showed advanced tabetic degeneration. The pia was the seat of a marked round-cell infiltration, slightly more marked around the anterior and posterior portions of the cord. There was some thickening of the walls of the vessels, the vessel lumen was narrowed, and the walls infiltrated with round cells. The veins showed infiltration of the outer walls in places. The anterior roots were intact, while the posterior roots showed marked degeneration and infiltration with round cells. There was border degeneration of the cord. There was a slight hemorrhage into the pons in the pyramidal tracts, apparently recent. There was evidently a syphilitic meningitis, with the typical blood-vessel disease found in this condition. Examination of the pia of the paracentral region showed thickening, vascular disease, and round-cell infiltration of connective-tissue type.

Six radicular nerves were examined, four of which included the ganglia. In one of the nerves there was slight degeneration by the Weigert method observed at the ganglion, without round-cell infiltration. More centralward there was observed slight degeneration in the posterior roots (Weigert), but no round-cell infiltration whatever. In the second nerve the posterior roots were markedly, the anterior roots slightly, degenerated. There was some perineural involvement, but no epi- or endoneuritis. The perineurium was thickened, but no round-cell infiltration was observed.

In the third radicular nerve studied the posterior root was intensely degenerated just as it left the ganglion, but there was no evidence of round-cell infiltration. In the perineurium there was slight infiltration. The anterior roots were intact. Centralward the round-cell infiltration of the perineurium was marked, and the epineurium was implicated, and this later involved both the anterior and posterior roots, when the endoneurium was diseased. The fourth radicular nerve showed no round-cell infiltration or degeneration of the roots. There was not very much of this root obtained and studied, and it was probably one of the dorsal roots..

In the fifth nerve there was no round-cell infiltration, though slight degeneration of the posterior roots by the Weigert method was observed. Otherwise the nerve stained well. In the sixth nerve the posterior roots were degenerated near the ganglion, but there was no round-cell infiltration. More centralward there was round-cell infiltration of the endo- and epineurium, and the posterior roots were found degenerated.

Case 14. — H. G., aged sixty-five. Family and previous histories negative. The symptoms were absent reflexes, sphincter involvement, lancinating pains, ataxia in the arms and legs, lost tendon reflexes in the legs, Romberg's sign, irregular pupils reacting to accommodation and sluggishly to light.

In the cervical region of the cord the anterior and posterior roots were intact, and the posterior columns were only slightly involved. In the thoracic region there was a border degeneration and degeneration confined to the middle and dorsal root zones in the lateral portion. The posterior roots were slightly degenerated; the anterior roots remained intact. There was only slight involvement of the central root zone. There was no infiltration of the pia in the cervical region, though it was thickened, and this was true of the other levels of the cord. The blood vessels were thickened, and some round-cell infiltration of the veins was observed. A small hemorrhage was found in the gray matter of the cortex. The pia of the cortex was thickened, and there was a sparse round-cell infiltration of connective-tissue type.

Four radicular nerves were studied, in one of which the ganglion was present. In the first nerve the anterior roots were intact, but the posterior roots showed intense degeneration. There was a hemorrhagic area of softening in the posterior roots, and the perineurium around the posterior roots was slightly infiltrated. More centralward there was some endoneuritis, but no round-cell infiltration of the endoneurium at this place. Still more centralward the epineurium was infiltrated, giving rise to degeneration of the fibers near the cell infiltration. Still more centralward the anterior roots were found slightly degenerated, which later became marked. In the second nerve studied the endoneuritis was intense, while the fibers stained fairly well by the Weigert method in the posterior roots, the anterior roots probably being intact. There was slight epineuritis, but no involvement of the nerve bundles. More centralward endoneuritis was observed without degeneration. Later there was intense degeneration of the fibers with marked round-cell infiltration.

In the third radicular nerve studied there was, near the ganglion, no degeneration by Weigert, and no peri-, epi-, or endoneuritis. More centralward, however, slight degeneration of the posterior roots was observed, while the anterior roots were intact. Perineuritis and round-cell infiltration were observed without epi- or endoneuritis; then, more centralward, there was slight degeneration of the posterior roots, with the anterior roots intact. Still further centralward there was intense degeneration of the posterior roots, and hemorrhage into the posterior roots with marked endoneuritis was present, though no epineuritis, but some perineuritis, showing that endoneuritis may exist without epineuritis. Finally, there was space formation, that is to say, softening due to hemorrhage into the nerve.

In the fourth nerve examined there was found posterior-root degeneration without round-cell infiltration of the epi- or endoneurium. More centralward there was observed degeneration of the anterior and posterior roots, more marked in the latter, without peri-, epi-, or endoneuritis. Still nearer the center there was endoneuritis with round-cell infiltration of the posterior roots, without destruction of the nerve elements, however. There was slight perineuritis, but no epineuritis. Later the perineuritis became intense. The blood vessels were not markedly diseased, though there was some hyalin degeneration of the walls.

In this case there was no marked round-cell infiltration of the meninges or radicular nerves. The cells in the radicular nerves, and the round-cell infiltration which was present, showed connective-tissue cells especially, though there were some lymphoid cells as well.

Case 15 (reported by Spiller, Syringomyelia and Tabes, Journal of Med. Research, Vol. 18, March, 1908). — J. S., aged sixty. No history of syphilis. Romberg's symptom, lancinating pains, ataxia of the hands, sensory disturbances, atrophy of the thenar and hypothenar eminences of the hands, lost reflexes in the arms and legs, Babinski on both sides, pupils reacting sluggishly to light, and promptly to convergence and accommodation, constituted the main symptoms. The symptoms were of thirty-one years' duration.

Examination of the cord showed typical degeneration of tabes, associated with syringomyelia. The pia showed infiltration of cells of the connective-tissue type, the infiltration being evenly distributed, and not more intense around the roots. The veins showed round-cell infiltration of the walls, more pronounced around the syringomyelic areas. The arteries appeared to be intact. The round cells consisted of the proliferative type of connective-tissue cells. Examination of the pia in the paracentral region showed thickening of the pia with slight round-cell infiltration of connective-tissue type.

Two radicular nerves, including one ganglion, were examined. The anterior and posterior roots were both degenerated slightly at the ganglion, the degeneration of the posterior roots becoming more intense centralward. There was no round-cell infiltration at the ganglia. More centralward the perineurium was involved, with slight round-cell infiltration, but no true implication of the endoneurium was noted. Nearer the center still, however, the round-cell infiltration invaded the endoneurium in the posterior roots, but not in the anterior roots. There was also epi- and perineuritis. In the second radicular nerve studied the conclusions were not very definite. There was slight degeneration of the anterior and posterior roots, but no round-cell infiltration.

The blood vessels in the radicular nerves were not diseased, and the round-cell infiltration was slightly more intense than in the meninges, though intense in both places. The round cells were of the proliferative connective-tissue type.

Case 16. — S., admitted to the Philadelphia Home for Incurables, March 12, 1900. Family and previous histories negative. Lancinating pains; Argyll-Robertson pupil; absent knee jerks; Charcot's joint; incontinence of urine; sensory disturbances; bedfast.

Typical advanced degeneration of the posterior columns, more marked in the sacral and lumbar regions. The anterior roots were slightly degenerated; the posterior roots markedly so. The pia was uniformly thickened at all levels of the cord studied, but there was no round-cell infiltration anywhere. The blood vessels were thickened, but did not show any infiltration of the walls. The pia of the paracentral region was thickened and moderately infiltrated with round cells of the connective-tissue type. The blood vessels were thickened but the walls showed no cellular infiltration.

Study of the radicular nerve of the tenth thoracic vertebra of the sensory root showed degeneration by the Weigert method, extending as far as the ganglia, but there was no round-cell infiltration. The motor portion of the root was intact.

Case 17. — S., admitted to the Philadelphia Home for Incurables with a diagnosis of locomotor ataxia. No history of this case could be found on the records, and the symptoms were not recorded.

Pathologically the following conditions were found: Typical tabetic degeneration of the posterior columns; degeneration in the posterior roots, and slight degeneration in the anterior roots. The pia was thickened throughout the cord, but no round-cell infiltration was found. The blood vessels were thickened slightly, but there was no infiltration of the walls. The pia of the paracentral region was thickened and moderately infiltrated with connective-tissue cells. The blood vessels were thickened, but the walls were not infiltrated.

Two radicular nerves were studied; in one, transverse sections, and in the other longitudinal sections were made. In both of these roots by the Weigert stain there was intense degeneration extending as far as the ganglion, but nowhere could be seen any round-cell infiltration except in the perineurium, where there was slight round-cell infiltration only. This did not extend to any of the other coverings of the nerve, however, at any level studied, and these roots did not present the appearance described by Nageotte. The sensory root was alone degenerated, the motor root staining well.

Case 18.—Case of cerebrospinal syphilis. T.L., aged fifty-four, was admitted to the Philadelphia General Hospital February 4, 1901. The family and previous histories were negative. There was no history of syphilis. Two years previous to admission, following an attack of unconsciousness, there was weakness of the legs which progressed to total paralysis over several months. There was then some return of power which failed in the six weeks before admission. His hearing was impaired for five or six years, but twenty months previous to admission he became very deaf in one week's time. He was subject to attacks of unconsciousness of one-minute duration, beginning with vertigo, and he suffered from intermittent headaches for two years. Vision was affected since the first attack of unconsciousness. There was a history of diplopia.

The symptoms consisted in wasting of the legs, especially the thighs; weakness of both legs and a sense of numbness in the legs with lowered sensation to pain and heat.

Knee jerks were absent on both sides; the Babinski phenomenon was present on both sides; the biceps jerks in the arm were present, and the triceps jerk absent on both sides. There was no ataxia in the arms. The pupils reacted slowly to light and accommodation.

When examined by Dr. Spiller on June 10, 1901, four months later, the atrophy of the lower limbs was very marked.

Power was retained in all muscles, but diminished. The knee jerks were entirely lost on both sides. There was no ankle jerk on either side. The plantar and cutaneous reflexes were exaggerated on both sides. Tactile sense was present everywhere. Pin-point was very sharply felt, and probably hyperesthetic. There was fairly good motor power in the upper limbs which were not much atrophied. The dynamometer registered on the right twenty-two; on the left thirty-eight.

Upon voluntary motion of the upper limbs a fine tremor was observed probably the result of weakness. The interossei muscles were probably atrophied. The pupils were unequal, the right smaller than the left, and irregular in outline, and contracted to light, but the action was slow. Contraction in convergence was very prompt. Station was very uncertain, but he was able to stand with the feet wide apart. The urine and feces were incontinent.

Examination by Dr. Pierce on April 13, 1901, showed reactions of degeneration in the anterior tibial group of muscles, and in the gastroenemic group in the right leg. Examination of the eyes by Dr. Hansel showed exophthalmus; a tendency to divergence with slow droop of the lid on downward movement of the eyeball; complete movements of the eyeballs, and clear media.

The spinal cord in the cervical region showed degeneration of the anterior and posterior roots, being more intense in the latter. There was, however, no degeneration in the intramedullary portion of these roots. Border degeneration was present, especially in the posterior columns around the most posterior portion of the longitudinal fissure. The posterior columns were diffusely degenerated in the posteromedian portions of both sides, but not resembling in any way a tabetic lesion. There was present a widespread perivascular round-cell infiltration throughout the white matter of the entire cord, especially adjacent to the pial indentations. Round-cell infiltration of the walls of the vessels of the pia and of the roots, and around the roots, was observed. Practically the same condition was found in the thoracic region, and in the lumbar region. The border degeneration in the lumbar cord was principally lateral, the posterior columns being slightly involved, if at all. The anterior and posterior roots were markedly degenerated. The round-cell infiltration consisted of mononuclear cells, as well as cells of the connective-tissue type.

Four radicular nerves were studied, in one of which the ganglion was included. There was peri- and endoneuritis present in one root, without any degeneration demonstrable by the Weigert method. In a second nerve there was degeneration by the Weigert method, but it was very slight and entirely out of proportion to the degree of cellular infiltration.

In the third nerve the perineuritis was marked, while the round-cell infiltration of the connective tissue within the bundles was slight. The perineuritis was marked in the posterior roots, and involved the endoneurium. The axis cylinders were swollen, but did not show degeneration by Weigert. The epineurium was markedly infiltrated. A little further away from the ganglion there was degeneration in the posterior roots by the Weigert method, and still further away some fasciculi, and some parts of other fasciculi in the posterior roots showed some degeneration. This was also present in the anterior roots, but was less marked. The degeneration in the fasciculi, studied by the Weigert method, did not seem at all in proportion to the degree of round-cell infiltration of the covering of the nerve bundles. Still further away from the ganglion there was marked peri- and endoneuritis, with preservation of the fibers, as seen by the Weigert method, except one bundle which was greatly degenerated, strongly contrasting with the healthy fibers immediately adjoining it, both of which were surrounded by round-cell infiltration. Further on there was localized degeneration of fibers, apparently due to round-cell infiltration of the perineurium.

The blood vessels of the radicular nerves showed marked round-cell infiltration of the walls. The degeneration of the roots near the cord was greater than in the radicular nerves.

SUMMARY.—Twenty-five radicular nerves from eight cases of tabes dorsalis and four radicular nerves from a case of syphilis were studied. In the eight cases of tabes studied eighteen radicular nerves were cut in serial section from the ganglion to the dura; in the remaining seven radicular nerves the ganglion had unfortunately been removed at autopsy, and only parts of the nerves could be studied. Of the four roots studied from the case of syphilis in only one was the ganglion present.

The lesion described by Nageotte was present in six cases, although very slight and insignificant in one case. It was absent in two cases. That is to say, there was demonstrated in all of the six cases round-cell infiltration of the radicular nerves involving one or more of the coverings of the nerve, namely, the peri-, epi- and endoneurium. This was traced as far as the ganglion in the case of syphilis, and in two cases of tabes (Nos. 11 and 14). In these two cases the degeneration of the fibers, by the Weigert method, was also observed.

Hemorrhagic areas of softening were seen in three cases, causing extensive destruction of the posterior roots (Nos. 12, 13, and 15). In two of the cases the symptoms were of long duration and in one had lasted over three years.

In one case (No. 14) two nerves studied showed degeneration by the Weigert method without round-cell infiltration of the peri-, epi- or endoneurium, except in one root where there was a slight perineuritis. In other roots in the same case round-cell infiltration and areas of softening were observed.

The degeneration of the roots extended toward the ganglion beyond the focus of cellular infiltration in some instances (Nos. 12, 13, 15). In one case (No. 15) endoneuritis was observed unassociated with peri- or epineuritis. In one case (No. 13) showing extensive cellular infiltration which extended as far as the ganglion, there was only slight degeneration by the Weigert method. The symptoms were of four years' duration in this case. Degeneration extended into the ganglion in seven cases (Weigert) (Nos. 11, 12, 13, 14, 7, 16, and 17). In two cases (Nos. 16 and 17) the radicular nerves showed degeneration by the Weigert method, but there was no round-cell infiltration observed either of the peri-, epi- or endoneurium.

The round-cell infiltration was composed of small round cells closely packed together, and in most instances it was marked in degree. The nerve fibers showed degeneration by the Weigert method in those places where the round-cell infiltration was seen.

In the syphilitic case (18) the appearances were identical with those in the cases of tabes. By the Weigert method, however, the degeneration was slight only, and out of proportion to the round-cell infiltration, which was intense.

The anterior roots were slightly degenerated in three cases (Nos. 14, 15, and 7), but intact in four cases (Nos. 12, 13, 16, and 17).

The cortex was studied in sixteen cases, all showing round-cell infiltration of connective-tissue type. In one case some plasma cells were also present; in another, polynuclear cells; and in a third case mononuclear leucocytes as well.

The meninges of the cord, studied in seventeen cases, showed pure connective-tissue type of cells in nine cases. In three cases mononuclear lymphocytes and connective-tissue cells were observed. In four cases no infiltration was noted, and in one there was only a very slight round-cell infiltration, although the pia was thickened in these cases.

The round-cell infiltration showed no predilection for position, except in two cases where the infiltration was more marked anteriorly and posteriorly, and in the longitudinal fissures.

In eight cases (Nos. 1, 6, 10, 11, 12, 13, 15, 9) the infiltration resembled that of syphilis, and was associated with changes in the blood vessels consisting of thickened walls and in some cases obliteration of the lumen and endarteritis.

In the eight cases of tabes in which the radicular nerves were studied, four presented the characteristic round-cell infiltration of syphilis, and two others were probably also syphilitic, although the infiltration was not absolutely typical.

Nageotte[1,2,3] was the first to describe neuritis of the radicular nerves in tabes, and he believed that it was the cause of the spinal cord changes in this disease. He claimed that syphilitic meningomyelitis was present in all cases of tabes, and that this was the immediate cause of the neuritis of the radicular nerves. Peri-, epi- and endoneuritis caused a local atrophy at the site of the inflammatory focus. The cellular infiltration consisted of fixed cells, lymphocytes, and a few epithelial cells. According to Nageotte the neuritis usually implicates the nerve at the middle third of the posterior roots, being situated a little nearer the cord in the anterior roots. The atrophy begins in that part of the nerve which is furthest removed from the ganglion, spreading toward the center. The regeneration occurs in the anterior roots, but is not seen in the central stump of the posterior roots, because the endoneuritis spreads downward into the ganglion, and destroys this portion of the fiber. The lesion is a transverse interstitial radicular neuritis of specific origin which is derived from a chronic specific meningomyelitis.

A late atrophy is observed which affects certain kinds of fibers in each root, and which is irregularly distributed to the different roots of the cord, and different fasciculi of each root. According to Nageotte the pia of the cord is thickened and infiltrated, the lesion predominating posteriorly. The thickening of the pia is due to hypertrophy of connective-tissue fibers, and the infiltration is largely lymphocytic, with some epithelial, plasma, and pōlynuclear cells. He claims that the vessels are always altered, particularly the veins.

The radicular nerve * lesion, he stated, is not constant in syphilitic meningitis, which explains why the latter does not always cause tabes. Radicular neuritis is, however, constant in tabes, he claims.

Objections to Nageotte's theory have been raised by Orr[4,5] and Rows, who have observed degeneration of the posterior columns without lesions of the extra-medullary

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* The radicular nerve is that part of the root which lies between the point at which it arrives at the dura, and that at which the posterior root reaches the ganglion.

course of the posterior roots, and they claim that degeneration of the intramedullary portion of the anterior roots which accompanies so often degeneration of the sensory fibers is the strongest argument against Nageotte's theory, and shows that a neuritic process in the extramedullary portion of the sensory and motor nerve is not essential. Moreover, they claim that the lesion is not a constant one, which is borne out by my observations, and that it is exceeded by the intramedullary lesion, and is different in character. They believe that the sensory fibers have been attacked in their intramedullary course by some toxin and at that point where they enter the cord the noxious agents cause degeneration which spreads centrifugally. Wallerian degeneration, they believe, spreads centrifugally from the point of injury. They quote the work of Erb, Tizzoni, Mönkeberg, Neumann, and Bethe in support of this view. This opinion is contrary to that held by Nageotte, who claimed that this degeneration began at the distal end of the fiber, and spread backward toward the trophic center.

That this lesion is not constant has been shown by Oppenheim,[6] who found it absent in two recent cases, and only present in one old case; and by Marinesco[7] who demonstrated it in only three cases out of five. Nor did Redlich[8] see that there was any parallel between tranverse neuritis and degeneration of the posterior cord. He stated that it was lacking in tabes at the beginning, and only apparent with difficulty later.

Thomas[9] and Hauser also declared that the lesion was not constant. While they found lesions comparable to those found by Nageotte, they did not believe that they were of capital importance in the process of degeneration of the roots. In meningo-myelitis syphilitica they claim the posterior roots are plunged into intense pachymeningitis without being implicated, and believe that the lesions of the meninges of the roots are episodic. They refuse to admit that the parenchymatous lesions are exclusively dependent on the connective-tissue lesions, not alone because they are inconstant and not in proportion to the atrophy of the roots, but because they do not seem to appertain properly to tabes. Moreover, meningitis is sometimes very intense around the anterior roots without implicating them. They found the nerve fibers implicated in traversing the morbid focus sometimes, and looked upon this as accidental and not the rule. If Nageotte's theory is correct they would expect to find atrophy of the radicular fibers in the interior of the ganglion as a retrograde process. They could not see that this process could produce itself if the radicular fibers were not profoundly degenerated in the primary focus. They have seen an atrophy slight in the posterior roots remain slight in the radicular nerve as far as the ganglion.

That this lesion is not characteristic of tabes seems proven by the findings of Massary,[10] who found it present in cases dying of tuberculosis, meningitis, icterus, pulmonary congestion, cerebral hemorrhage, typhoid fever in a child, and pleurisy, and who felt justified in the conclusion that the lesion was without significance.

Cassirer and Strauss[11] cite, in opposition to Nageotte's theory, cases of incipient tabes associated with syphilitic ulcer between the esophagus and larynx, orchitis syphilitica, rachitis, and thickening of the bones, cases which showed, beyond a slight pial thickening, no meningitis. They believed that this did not support the view that there was always a meningeal origin for the degeneration of the posterior columns.

Marinesco found the fibers in the cord more implicated than in the posterior roots, and in the posterior roots more than the intraganglion fibers, showing, he thought, that the lesion began in the cord and not in the roots. Marinesco and Minea[12] stated that proof of a parallel lesion affecting all the tracts of the central white matter, similar to that of the starting point of the lesion, is of great importance, for it shows first that the lesion of tabes consists of a primary atrophy of the central branch of the sensory system; second, that it is not of interstitial origin, as Obersteiner, Redlich, and Nageotte claim, but of parenchymatous origin. If due to compression of the nerves, or transverse radicular neuritis, it would be impossible to comprehend why this lesion involved at the beginning the intraganglion fibers. These should remain intact, as in compression from section of the nerve, when the peripheral end degenerates in spite of the fact that the central end remains undegenerated.

My own observations are evidence against Nageotte's views. For example, in the case of syphilis the radicular nerve lesion, which is described by Nageotte, was present, but there was no tabetic degeneration of the posterior columns. Moreover, in two of the cases there was degeneration of the radicular nerves without round-cell infiltration of the peri-, epi- and endoneurium. Furthermore, the degree of round-cell infiltration was not proportional to the degeneration in the roots as a rule. That is to say, the degeneration is intense when there is slight, as well as intense, round-cell infiltration, or with absence of this lesion.

The disproportion between the round-cell infiltration and the degeneration in the radicular nerves is not an argument against his theory, Nageotte believes, for he asks: What parenchymatous lesion *is* always proportional to the interstitial lesion? He believes that individual resistance comes into play, and calls attention to the fact that the syphilitic infection is not always uniform, and the lesions are variable. But the fact that neuritis of the radicular nerves is seen in other conditions as described by Massary, and as observed in my case of syphilis of the nervous system, is, in my opinion, one of the strongest arguments against Nageotte's theory.

MENINGITIS. — The presence of meningitis in tabes dorsalis has been frequently described. Whether or not it is constantly present is still open to discussion. It was present in thirteen of the seventeen cases in my series. In four of the seventeen cases it was not demonstrated, in any of the sections studied, in the meninges of the cord, although the pia was thickened, but in the meninges of the paracentral region in these four cases there was some infiltration. The round-cell infiltration did not differ greatly from the round-cell infiltration of syphilis. The process was evidently a chronic one, the cells being of a connective-tissue type in most instances. In three cases the connective-tissue cells were associated with mononuclear leucocytes. The typical specific appearance of the meninges was demonstrated in eight cases, including the characteristic blood-vessel change of this disease. There was no predilection as to the situation of this round-cell infiltration, which predilection, according to Marie and Guillaine,[13] is for the posterior meninges. They also state that the lesion is less pronounced or absent in the sacral region. This was not confirmed in my cases, as the meningitis was present in the sacral region in some cases. Lepine[14] claimed that the primary sclerosis localized the meningitis, while Vulpian,[15] on the contrary, believed that there was no uniformity in the meningeal lesion.

In my cases the impression given was that the meningitis was not sufficiently intense to be considered an important factor in the causation of the lesion that was found in the roots or the posterior columns. It was not constantly present, being practically absent in four cases. This observation coincides with that of Redlich, who did not believe the meningitis was a constant finding in tabes. He claimed that there are some cases in which it was not present, although there was thickening of the pia, and he was of the opinion that the primary posterior sclerosis localized the meningitis. Leyden[16] regarded it also as secondary.

On the other hand, Lang[17] believed that meningitis was a constant finding, and according to Arndt[18] the meningitis was the starting point of the tabetic disease, while Türk[19] long ago called attention to the meningeal change in disease of the posterior columns.

The evidence in my cases points to the conclusion of some authorities that the meningitis as well as the radicular changes are a part-symptom, rather than the cause, of the tabetic degeneration. Typical cases of tabes clinically have been cited supporting this view, in which there has been associated pathologically syphilis of the nervous system and other organs. Examples of these are Schittenhelm's case[20] of incipient tabes in cerebrospinal syphilis; Sach's case[21] of undoubted tabes with specific round-cell infiltration of the pia, and blood-vessel change; Nonne's three cases[22] complicated by gumma of the cerebrum and one of incipient tabes and specific spinal meningitis; Cassirer and Strauss's cases[11] of incipient tabes with syphilis of the other organs; Fraenkel's case[23] of tabes and specific arachnitis and leptomeningitis obliterans; Hoffman's case[24] of specific meningitis and diseased blood vessels; Pick's case[25] of classical tabes and chronic specific meningitis; Sydney's case[26] of vascular change of the spinal cord, and similar cases of Adrian,[27] Miner,[28] and Dinkler.[29]

The cases of pseudotabes in which there is a syphilitic meningo-myelitis without tabetic change in the posterior columns, seems to me to be evidence in opposition to Nageotte's views. I refer to the cases described by Giuffre and Mirto,[30] Lamy,[31] Maplain,[32] Eisenlohr,[33] Ewald,[34] Oppenheim,[35] Wolff,[36] and others, in which there were present atypical symptoms of tabes clinically, and lesions characteristic of cerebrospinal syphilis pathologically.

If meningitis is not constantly found in all stages of the tabetic process, it is certainly a *frequent* finding, and probably present at some time in the progress of the disease. In the four cases in my series in which the round-cell infiltration was not observed, the pia was thickened. However, border or *Rand* degeneration was found in eight of my cases (Nos. 1, 5, 3, 10, 11, 12, 13, and 16). In one of the cases in which there was no meningitis demonstrable, the *Rand* degeneration was found, and probably also in a second case, indicating that probably at some stage of the pathological process meningitis had been present. *Rand* degeneration has been already described by Pick, Edens,[37] and Minor in cases of tabes complicated by specific meningitis. The question whether the meningitis corresponds to the intensity of the process in the spinal cord has been answered in the negative by my cases. There was no evidence that the meningeal lesion was more intense posteriorly than elsewhere.

Is the meningitis present in old as well as in recent cases, and does the infiltration differ in character in the old and recent cases? This question must be answered by future observations.

In two advanced cases in my series the round-cell infiltration of the meninges was very intense, but in other cases where the tabetic degeneration was intense and advanced, the meningitis was less marked, and out of proportion to the systemic disease.

It has already been shown that the pathological appearance of tabes when the other organs are syphilitic, does not differ from those when the organs are not syphilitic (Cassirer and Strauss). This was true in one case of mine in which there was syphilitic myelitis plus tabes (No. 13). It is also clear that the pseudotabes, due to syphilitic myelitis, has a different anatomical basis.

There is no doubt that syphilis, either of the organs, skin, bones, or central nervous system and tabes may occur as independent processes at the same time and in the same individual.

CONCLUSIONS.

1. The radicular nerve lesion is not constant in tabes dorsalis.

2. Meningitis is not constant in all stages of tabes dorsalis.

3. Lesions of the posterior columns in cases of tabes dorsalis complicated by syphilis do not differ from those found in cases in which there is no evidence of syphilitic lesion.

4. Syphilitic meningitis implicating the radicular nerves in cerebrospinal syphilis may exist without causing the characteristic tabetic spinal-cord lesion.

5. The meningitis in some cases of tabes is typically specific in its appearance, but differs in degree in some and is not present in all cases.

6. The cerebral meninges were infiltrated with round cells in all the cases studied.

7. *Rand* or border degeneration is present in a certain proportion of the cases. This is an observation that has already been made, but not emphasized, in the literature.

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DESCRIPTION OF PLATES.

Plate XVIII.

Fig. 1. — Shows intense degeneration of the posterior root of the radicular nerve and very slight degeneration of the anterior root (Weigert stain).

Fig. 2. — Shows degeneration of the sensory root extending as far as the ganglion. The anterior root is intact.

Plate XIX.

Fig. 3. — Shows hemorrhagic softening of the posterior root of the radicular nerve and intact anterior roots.

Fig. 4. — Round-cell infiltration of sensory root of a radicular nerve from a case of meningo-myelitis (Hæmalum and acid fuchsin).

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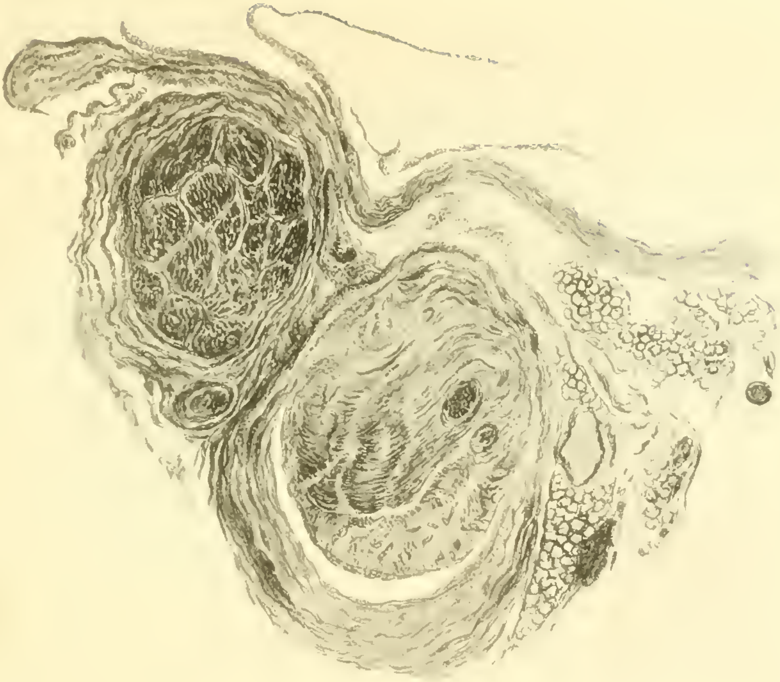

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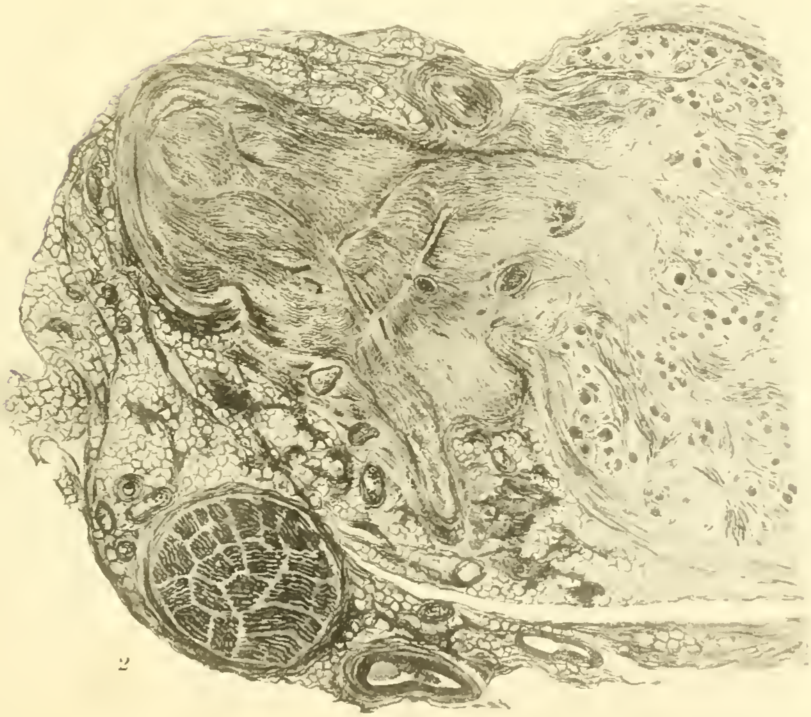

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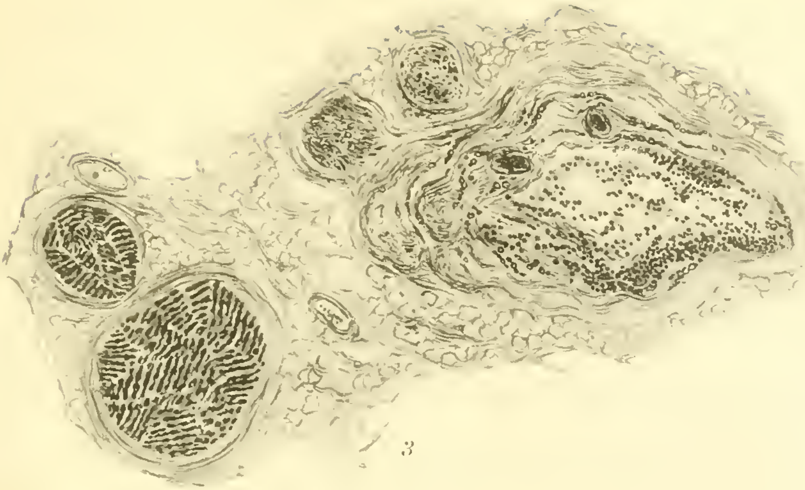
Rhein

Disease of the Radicular Nerves



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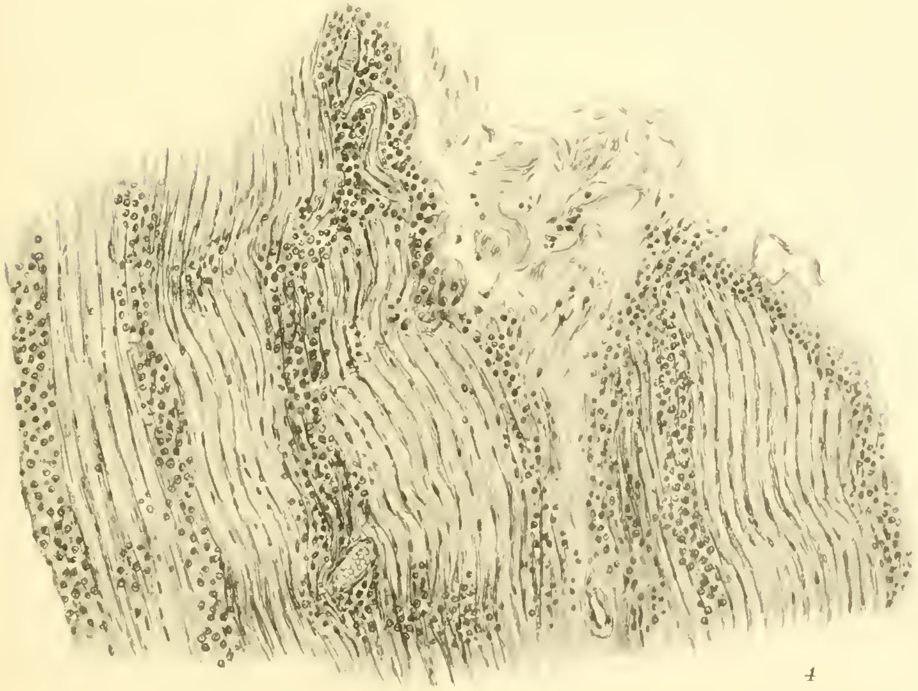

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Rhein

Disease of the Radicular Nerves

HYSTERIA, WHAT IT IS AND WHAT IT IS NOT.*

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Hysteria, a favored topic with medical writers since the time of the father of medicine, has again come to the front under the stimulating influence of Babinski, who has attracted the attention of the neurological world to this old subject, as Marie, a few years since, revived interest in aphasia. In the case both of aphasia and hysteria, views regarded as classical if not irrefragable have been attacked with skill and audacity. The profession of medicine owes much both to Marie and to Babinski, even if it is not prepared to give universal acquiescence to their views. Much that Babinski[1] has advanced should be received without demur. One thing of great value which has come out of his studies and writings has been the more exact delimitation of the definition of hysteria, even if the bounds set by him are not accepted in their entirety. He has done for hysteria what our distinguished American colleague, Dr. Dana, has accomplished for another nervous disorder so often discussed with hysteria and not infrequently combined with it in a clinical case, in his paper on "The Partial Passing of Neurasthenia." It might be said also that Dana,[2] after the fashion of Babinski, and almost contemporaneously with the latter's first general presentation of his conception, has set limits to hysteria which are not very different from those of Babinski, although expressed in somewhat different terms.

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* Read at the sixty-fifth annual meeting of the American Medico-Psychological Association, Atlantic City, N. J., June 1-4, 1909.

[1] Ma Conception de l'Hystérie et de l'Hypnotisme (Pithiatisme). Conference faite à la Société de l'Internat des Hopitaux de Paris, June 28, 1906.

Émotion, Suggestion et Hystérie. Extraits des comptes rendus de la Société de Neurologie de Paris, July 4, 1907.

Instabilité Hystérique (pithiatique) des membres et du tronc. Extraits des comptes rendus de la Société de Neurologie de Paris, March 5, 1908.

Démembrement de l'Hystérie Traditionnelle, Pithiatisme, 1909.

The tendency in the past has been to regard too many clinical phenomena as hysterical—to assign to hysteria every sort of nervous affection for which no other abiding place could be found. In a few respects Babinski has perhaps narrowed too much the conception of hysteria.

The definition of hysteria has been often essayed. Reference to a few of these definitions might here be made. In doing this I shall refrain from recalling any of my own efforts in this direction, as these with more experience have become more or less unsatisfactory. Starr, in his recently published work on "Nervous Diseases, Organic and Functional" (second edition, 1907), speaks of it as "a functional nervous condition characterized by a permanent mental state, which may be termed the hysterical temperament, and by sudden temporary attacks, of mental or emotional or physical kind." A definition like this, although necessarily imperfect, answers well enough for general descriptive purposes. The inclusion by Starr of the hysterical temperament as a permanent condition is important, as indicating that always underlying hysterical phenomena is a constitutional predisposition.

Dana, in his article on "The Limitation of the Term Hysteria," in his usual terse and thoughtful manner, has given a definition which appeals to those who have dealt practically with the disease when he says that hysteria "is a morbid mental condition in which ideas or emotional states seriously and unwittingly control the body and produce more or less permanent and objective morbid states." In this definition he introduces an etiological factor largely disregarded by Babinski, and insists with correctness on the possible greater or less permanence of the phenomena of the disease, in this respect also differing somewhat from the French neurologist.

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[2] "The Partial Passing of Neurasthenia." Boston Medical and Surgical Journal, March 31, 1904.

"The Limitation of the Term Hysteria, with a Consideration of the Nature of Hysteria and Certain Allied Psychoses." Journal of Abnormal Psychology, February, 1907.

The etiological and therapeutic definition of Babinski is that now most attracting attention, but this, like all others, is far from being completely satisfactory. While it is impossible to state what the transient anatomical substrata of hysterical phenomena are, in every definition which deals with the supposed nature of hysteria, some consideration must be given to this matter, as well as to the question of inherited tendency. An affection which presents such definite symptoms and signs as those which are recognized universally as hysterical must have beneath it a change of some sort in the nervous system, even though this is of a more or less transitory character. The disease is functional in the sense that this change is not necessarily permanent, and healthy conditions can often, but by no means always, be readily restored; but disease it is, whatever its nature and however short or long its duration. What the change in the nervous system is, the most extensive observation and investigation have not enabled us to learn, but the same may be said of other so-called mental and nervous disorders which are everywhere appreciated as real diseases—of melancholia, mania, the neuralgias and chorea, for instance. The speculations advanced like those of cortical vasomotor spasm, of toxemia, and of neuronal disseverance, are all open to the objection that they cannot be proved and are not on the basis of analogy truly explanatory of the phenomena.

Objections of another sort may be urged to the psychological definitions which discuss hysteria as a dissociation or disintegration of personality. This it probably is, but not much help is given to the student or the practitioner by such a definition unaccompanied by a consideration of both predisposing and exciting etiology, and of its generally recognized clinical phenomena. The disease is genuine, not simply imaginary or factitious, even if the conditions underlying it are not demonstrable by any known methods.

A partial error in the definitions of both Dana and Babinski is that sufficient emphasis is not laid upon the abortive or imperfectly developed forms of hysteria. These constitute a considerable percentage of the cases which should be really regarded as instances of hysteria, and no more reason exists for not recognizing them than for not believing in the abortive forms of other well-known diseases as, for instance, the *formes frustes* of exophthalmic goiter. These cases of hysteria in which hypesthesia, sometimes of light grade, moderate or slight impairment of motor power, choreiform movements, tremor, and other well-known phenomena are present, are as truly instances of the disease, although of less severity, than those observed in the rare disease described by Dana, in which gravely marked and more permanent symptoms and signs can be demonstrated. The same constitution or temperament usually underlies them, and they present the same or similar stigmata, these differing only in their intensity. That they are amenable to treatment is not an argument against their genuineness.

The main advantage of Babinski's definition that hysteria is a nervous disorder induced by suggestion and cured by persuasion, is that it concentrates attention upon what the framer of the definition believes are its great causative and curative agencies. It leaves the fundamental nature of the disorder unexplained, while presuming, of course, its psychic pathology.

Babinski includes in his list of hysterical stigmata, that is, in his series of phenomena which are capable of being produced by pure suggestion and cured by persuasion, " convulsive attacks; somnambulism; delirium; paralysis; divers contractures; tremors; cloreiform movements, usually rhythmical; disturbances of phonation and respiration; disorders of sensibility shown in anesthesia and hyperesthesia; sensorial anomalies; and disturbances of the bladder."

Speaking of what should be excluded from the hysterical syndrome, he tells us, " Suggestion cannot abolish or exaggerate tendon reflexes or cause disturbance of the pupillary reflexes or the cutaneous reflexes; at most, suggestion can only render examination of these phenomena difficult, or place obstacles in the way which might mislead a novice, but which an experienced neurologist would overcome; suggestion cannot induce vasomotor, secretory or trophic disorders, and it cannot alone induce hemorrhage, anuria, albuminuria or fever."

Let us turn aside for a moment to say a word about terminology. All things considered, it is better to retain the word hysteria unless some term unequivocally expressive can be substituted, and even Babinski has failed in this respect. In order to enforce his views he has coined the new term pithiatism and its derivative pithiatic from Greek words meaning "persuasion" and "curable." As he recognized, this new term refers to only one, although an important, feature of his definition. It does not indicate the idea of suggestion as a cause, although he regards suggestion as one of two factors of his definition. An earlier recommendation than that of Babinski, of a term to describe hysterical and hypnotic phenomena, was suggignoskism or suggestionism, but this term is as objectionable as pithiatism, and for a somewhat similar reason, namely, that it is not sufficiently descriptive.

Is it altogether philosophical to describe a disease by a term which is supposed to indicate the method of successfully treating it? Objections might be offered to this therapeutic-test method in terminology as in diagnosis. Quinine in malaria, mercury in syphilis are remedial or curative agencies. Should one, therefore, define these diseases, not as affections with special pathogenesis and well-known clinical phenomena, but in terms which indicate that they yield to certain medicinal remedies? No one but Babinski and his immediate following will dispute that hysterical affections will not always yield to methods of persuasion or education, unless these are supplemented by other measures calculated to improve the general nutrition of the patient and to counterbalance inherited predisposition. What neurologist of large experience has not seen, especially in his private work, cases of grave hysteria which have resisted all suggestive and persuasive efforts until measures of a physical character have been used? It is true it has been said that the so-called rest treatment, with its seclusion, massage and electricity, is only a special method of applying suggestion and persuasion. Abundant experience, however, shows that massage and electricity, and other physical measures, do much more than merely stimulate the hope and imagination of the patient. They improve nutrition by aiding digestion, assimilation and elimination. That they improve the quality of the blood has been shown by actual investigation. In many cases of hysteria and hysteroneurasthenia, and especially the latter, the physical measures employed play a large part, and sometimes a part which is little if at all inferior to the psychotherapy exerted through the personality of the physician. Both the psychic and physical methods have their appropriate places and proportions in the treatment of hysteria.

The differentiation of hysteria from organic disease is to be chiefly made in the first place by the presence of certain phenomena which are pathognomonic of the latter. It has often been said that hysteria may counterfeit almost any form of organic disease, but this is not a correct way of putting the matter. It should be rather said that hysteria may more or less imperfectly counterfeit many forms of organic disease of the nervous system. With regard to these affections about which difference of opinion is most likely to arise, usually the presence or absence of certain well-known symptoms or signs, among which are those which Babinski insists are not present in true hysteria, will be sufficient to settle the diagnosis.

This would appear at first sight to be merely saying that organic disease is to be distinguished by organic symptoms, and hysteria by those which do not belong in this category. The difficulty is that too often the mind of the diagnostician has been in doubt as to what are the intrinsic characteristics of these differing affections.

The most reliance in the differentiation of hysteria from organic disease is to be placed upon a study of the reflexes, and of the manner in which the symptom picture of hysteria usually is exhibited. With regard to the deep reflexes, such phenomena as foot clonus and the Babinski sign—extension of the toes and especially the great toe upon plantar stimulation—are the most important. I have long taught that persistent foot clonus is almost invariably indicative of the existence of organic disease, and especially of disease of the pyramidal system.

In the discussion of a case of hemiplegia with contractures and tremor in the paralytic limbs, presented at the meeting of the Philadelphia Neurological Society, February 22, 1897, the importance of persistent foot clonus was insisted upon as a sign of organic disease. Such clonus was present in this patient and was sufficient, with the full consideration of the other phenomena exhibited, to exclude the case from the hysterical category.[3] Reference was made in this discussion to other cases in which ankle clonus of the persistent type enforced the diagnosis of organic disease, and attention was called to the fact that in some of these cases hysterical epiphenomena were present.

[3] Journal of Nervous and Mental Disease, July, 1897, Vol. XXIV, No. 7.

That an abortive or pseudoclonus may be present in hysterical and hysteroneurasthenic cases must be admitted. Gowers has described this as a spurious clonus, and believes that it is due to a half voluntary contraction of the calf muscles. Although this is occasionally seen, it is much more frequently absent than present, and certainly is not to be referred to the same causation as the persistent clonus of organic disorders.

With regard to the Babinski reflex, I find myself fully in accord with the discoverer of this sign, and this in spite of the recent conversion of Van Gehuchten,[4] on the basis of an exceptional case, to the side of those who believe that in rare instances both the Babinski response and foot clonus may be present in grave hysteria.

Owing to the high position of Van Gehuchten it would seem worth while to refer to the paper in which he records his change of mind. After an interesting general discussion of the physiological and pathological reflexes, tendinous and cutaneous, Van Gehuchten says that he held to the same view as Babinski until he became doubtful of its validity by observing a case in which, after having made the diagnosis of organic disease because of the presence of these signs, he was led to change his diagnosis and his opinion because of their disappearance in a few days.

He formulates as his final judgment that exaggeration of the tendon reflexes, even going as far as foot clonus, while characteristic of a lesion of the corticospinal fibers, *may* nevertheless be exceptionally met with in hysterical paralysis, and that the Babinski sign is not *always* pathognomonic of a lesion of these fibers, in that the typical extension of the great toe may be observed, however exceptionally, in hysteria. In order to explain his single exceptional case, he reasons that functional disruption or temporary disablement of the corticospinal system of fibers may be sufficient to account for the presence of the clonus and the Babinski sign. He recalls cases observed by him and others in which the Babinski sign was present, and in which no degeneration or irritation of the pyramidal bundles existed or could have existed, cases, for example, of tumor of the brain outside of the motor system, and of intraspinal tumors compressing the cord and not giving rise to pyramidal degeneration.

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[4] Van Gehuchten, A. Le Nevraxe Recueil de nevrologie normale et pathologique, Vol. VIII, Louvain, Uystpruyst., 1906.

In spite of this unusual case and the more or less cogent reasoning of Van Gehuchten, my personal experience has been altogether in favor of the position of Babinski. I do not recall a single instance in which the Babinski sign and persistent foot clonus or either were present, and the case demonstrably not one of organic disease. In making the statement it must be remembered that toxemias, and alterations such as are sometimes found in syphilitic disease, may give rise to temporary disablement of the corticospinal system, but these should be ranked among organic cases.

In some of the cases of tumor in both brain and spinal cord in regions somewhat removed from the pyramidal tract, in which the signs under consideration are present, these may be explained by the pressure exerted by the lesions on this tract.

It is not true that both the deep and the cutaneous reflexes are never disturbed in hysteria. In hysterical cases, especially in those in which anesthesia or hypesthesia is a unilateral sensory phenomenon, a difference is not infrequently shown between the knee jerks of the two sides. Usually, but not always, the kick on the side in which sensation is retained is more marked than on the other side. I agree with Knapp,[6] who in a paper recently read before the American Neurological Association, held that there is often a difference both in the cutaneous and deep reflexes on the two sides in hysterical hemianesthesia and hysterical hemiplegia.

It is a matter of the commonest observation that the plantar flexion cannot be obtained, or only in very slight degree, in cases of anesthesia which involve the foot or feet. Here it may be said that the reflex response is conditioned by the impairment of sensibility; nevertheless this is hysterical, and not until it has disappeared does the normal response return. In not a few cases of typical hysteria without impairment of sensation in the lower extremities plantar flexion is unduly active.

With regard to disturbances of the pupillary reflexes, it must be noted that excellent observers have put on record cases of iridoplegia as hysterical in origin. These are probably instances of faulty observation or faulty interpretation. It is probable that a toxemia or a transitory lesion like that which is sometimes present in nervous syphilis may be the true explanation. A degree of anesthesia also may play some part in producing a pseudo-iridoplegia, but this is a false and not a true paralytic disturbance.

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[6] Knapp, P. C. Proceedings of the American Neurological Association meeting, held in New York, May 27, 28 and 29, 1909.

One of the methods of distinguishing an hysterical from an organic paralysis which is worthy of attention and favorable comment is that of Hoover.[6] This observer studied a normal individual recumbent in the dorsal position, and found that whenever one leg was voluntarily raised from the couch the heel of the other exerted complementary opposition by being dug or pressed into the couch. Similarly, in the case of an organic hemiplegia or hemiparesis, when the sound leg is raised the affected leg, in proportion to the power which remains in the member, exerts a similar complementary opposition. In the case of either pure malingering or an hysterical hemiplegia or paraparesis, this complementary opposition does not take place unless indeed the person investigated, being familiar with what is expected of him, executes the test through the exercise of his will. It is easy, however, according to Hoover, in most cases to determine the presence of this sign.

My experience with Hoover's test has been limited but confirmatory of its value. In the hysterical hemiplegic or hemiparetic the affected limb remains passive throughout the test.

For generations most writers on the subject of hysteria have given a place of first importance to emotion in the etiology of hysterical attacks. Illustrations of this statement could, of course, be adduced in large number, but to take only two from recent works of high rank, Dana, in his "Text-Book of Nervous Diseases and Psychiatry," says that "The most important single exciting factor is powerful emotion, particularly fear," and Starr, in his "Nervous Diseases, Organic and Functional," speaks of mental or emotional shock as the chief exciting cause. Many cases have been put on record of the sudden or rapid production of hysterical phenomena or attacks through or apparently through the influence of sudden emotions—cases of paralysis of one or more limbs, of anesthesia variously distributed, of severe and prolonged convulsive attacks, of delirium, aphonia and visceral disturbances, all recognized as of hysterical character.

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[6] Hoover, C. F. A New Sign for the Detection of Malingering and Functional Paresis of the Lower Extremities. Journal of the American Medical Association, August 20, 1908.

Babinski, in order that his iron-clad definition may not be impaired, finds it necessary to exclude emotion from the causes of hysteria, but to do this entirely is not easy, and it is here, as in some other matters, that his views of the subject show something less than impregnability. He is confronted, for instance, with the necessity of explaining away such a time-honored illustration of the power of emotion over hysterical symptoms and conditions as the sudden cure of hysterical paralysis by the fear of fire which unexpectedly threatens the life of the patient. "Let us," he says, " take another example, that of a woman afflicted with paralysis or with hemiplegia rebellious to all attempts at psychotherapy, who, seeing that the house is on fire, jumps out of bed, runs out, and thus is restored suddenly to health. In this case emotion indeed seems to be the cause of the cure, but on reflection this conclusion becomes a matter for discussion. It must not be forgotten that it is usual to say to such patients that they will one day get well suddenly under the influence of joy, terror or some other moral shock; and it may be presumed that such a prediction coming to mind at the moment of the conflagration exercised a psychotherapeutic influence."

It must strike everyone that an explanation such as this lacks somewhat in force or in ingenuousness. The simplest explanation which appeals to everyone is that it is the sudden and tremendous influence of the emotion of fear which produces the result. It is hardly necessary to invoke the theory that suggestions previously given act as the therapeutic agent. If suggestion plays any part, it is most likely that it is the suggestion of death or great bodily harm, which is suddenly called into being by fear.

Only one additional word need be said in this connection. An emotion capable of thus curing an hysterical paralysis would certainly be capable of producing it.

The hysterical nature of some vasomotor and secretory affections had up to the time of Babinski generally been accepted, and this chiefly because of their frequent association with other generally recognized hysterical phenomena such, for instance, as those enumerated by Babinski and by Dana. That these affections, among which may be enumerated blushing and pallor, abnormal perspiration and disorders of the alimentary canal, may be produced or exaggerated by suggestion, probably no one will deny. Babinski, however, and correctly in accordance with his dictum, asserts that they are not influenced, or at least not as much influenced, by persuasion as those phenomena which he regards as evidences of genuine hysteria, and therefore are not truly pithiatic. Of course, if it is admitted that only well-marked pithiatic symptoms are hysterical these conclusions must be accepted, but it is somewhat doubtful whether absolute acquiescence should be given to this view of the matter. Admitting the influence of emotion in the etiology of hysteria, no good reason would seem to exist for not including some vasomotor and secretory affections as phenomena, or at least epiphenomena of hysteria. Psychic influence plays a major part in their production and, as has been shown in some experiments upon animals and upon human beings, a like influence upon their disappearance or upon their non-appearance. It is not within the scope of this article to go into the question of the influence of emotional states on the alimentary canal and on vasomotor and secretory phenomena in general, but many observations, recent and remote, have shown the great importance of this influence, and have also shown that morbid phenomena, alimentary, cutaneous and visceral, are frequently observed among the hysterical. The problem may perhaps best be left as at present, not fully solved.

At the meeting of the Paris Neurological Society, held July 4, 1907, in reply to Ballet's query as to how he (Babinski) would explain the fact that the most characteristic manifestation of hysteria, the attack, is generally provoked by an emotion, Babinski spoke as follows:[7]

> Even admitting that apparently spontaneous hysterical attacks are always the result of emotion, it is no less true that attacks of this kind may be experimentally reproduced, and the form, intensity and duration may be varied at will; they are thus accidents capable of being provoked by suggestion, and, in consequence, should be ranged with hysteria, in

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[7] Loc. cit.

accordance with the definition which I have given of this nervous condition.

"Further, I think that the rôle of emotion in the genesis of hysterical crises has been exaggerated. Doubtless emotion alone may give rise to such trouble as the constriction of the throat, and the intellectual confusion, more or less pronounced, that is described as belonging to the hysterical attack; but are these not rather physiological reactions which any individual may present?

As far as concerns the most characteristic symptoms of the attack, the opisthotonos, the passional attitudes, etc., there is room to believe that these are less the consequence of emotion than of suggestion, or of imitation, which is a form of suggestion.

It appears to me also quite admissible that in certain cases the emotion, producing a psychic disturbance which diminishes the faculty of control, augments the suggestibility and constitutes a condition favorable to the display of the hysterical manifestations. These are, however, questions which we may discuss forever, for they lead to no definite solution.

But it is incontestable that so-called primary hysterical accidents, particularly the hysterical attacks, whatever be the part belonging to emotion in their genesis, may be reproduced rigorously by suggestion, and they are thus to be distinguished from the emotive pilomotor and vasomotor disturbances upon which I have addressed the society."

Is there not much begging of the question in this reply and explanation of Babinski? Would it not be better to frankly admit the real rôle of emotion in the causation of hysteria than to make the part which it plays indirect in order to escape from a difficult position? Whether emotion acts by direct or indirect suggestion, or in some other way, it plays a part usually in association with other causes, as physical injury, in the production of hysteria, especially of the grave type. To make emotion, however, the most frequent exciting cause of hysteria is as serious a mistake as to exclude it altogether.

It may be worth while, after discussing this question of the rôle of emotion in causation, to cite the views of Oppenheim as to emotional phenomena in the clinical history of hysteria from the last edition of his Lehrbuch der Nervenkrankheiten.[8]

"From all this we may conclude: The foundation of hysteria is an abnormal mental state. The anomalies concern in the first place the affective sphere. There exists a disparity between the intensity of the stimulus and the strength of the emotional reaction, mostly in the sense that the emotional excitability is exaggerated, and the stimulus-threshhold is depressed. To this is due the inconsequence, the incalculable character of the reaction, by which one and the same stimulus may leave the emotional sphere completely uninfluenced, or may set free powerful as well as qualitatively varying emotional states and emotional discharges. Disturbances also are noticeable in regard to the duration and stability of emotional excitation. On one occasion a feeling of dislike, awakened through practically one impression of an idea, may last an abnormally long time, and produce a mood which is morbid in its persistence; on another occasion the emotional process may be manifested with great inconstancy through abrupt, apparently unmotived oscillations. The 'hysterical capriciousness' (moral ataxia, Huchard) is certainly not a constant symptom, or one occurring in all cases, but in the majority of cases it is clearly marked. Doubtless also a pathological diminution of the emotional reaction ('emotional torpor') may exist, especially in certain cases of suffering."

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[8] Oppenheim, H. Lehrbuch der Nervenkrankheiten, Ed. 5, 1908, Vol. I, pp. 1202-1204.

To the members of this association the relation of hysteria to insanity is a topic of special interest. While no trained neurologist or alienist will make the mistake of confounding the well-known forms of insanity, such as melancholia, paranoia and general paresis with hysteria, even these affections in their lighter forms or earlier stages are occasionally set down as hysterical by the general physician. This is particularly true of hypochondriacal melancholia, neurasthenia sharing with hysteria in the confusion of diagnosis exhibited by the physician. It is, however, more especially from the group of mental phenomena which Janet has marshalled under the term psychasthenia that it is necessary to clearly differentiate hysteria.

This differentiation is really easy if one's conception of hysteria on the one hand and of psychasthenia on the other is perfectly clear. It need not, however, be a matter causing stupefaction, as Babinski suggests regarding a colleague, if some cases of the two affections seem to have analogies. The hysteric, although not frequently, may show obsessions of doubt or fear or other typical manifestations of psychasthenia, and the psychasthenic may, in rare cases, present genuine hysterical stigmata. Having in mind these rare occurrences the diagnosis of one of these now well-defined affections from the other is no longer, thanks to Janet, Babinski, Prince, Donley and many others, a task requiring unusual powers; nevertheless Babinski lays perhaps too much stress upon the values of his etiologico-therapeutic definition of hysteria in connection with the separation of these disorders. Certainly persuasion, in the sense of proper educational measures, plays an important part in the extremely rare cases in which the cure or great improvement of psychasthenia is possible. The most important point in the differentiation is the absence in pure psychasthenics of the recognized stigmata of hysteria. While, as indicated above, these may be associated symptoms or epiphenomena, on the whole the association or the superimposition is infrequent.

With regard to hysteria and neurasthenia the difficulties of diagnosis arise only in those cases which are poorly studied or in which the genuine symptoms of both affections are commingled. A tendency is shown by some to speak of a case as either neurasthenic or hysterical without due consideration. The symptomatology of neurasthenia is far more complex than that of pure hysteria, but the one condition which dominates the entire symptomatology is that which shows the existence of exhaustion of nerve cells. In this way only are to be explained the fatigue on moderate or slight exertion, mental or physical, the more or less isolated or the widely distributed paresthesia, the exaggerated reflexes, the sexual and vesical weakness, the epigastric pulsation, and the numerous other well-known symptoms of neurasthenia, varying according to the so-called types—cerebral, spinal, cerebrospinal, lithemic, sexual, angiopathic, etc.

Mettler,[9] who has written a valuable article contrasting hysteria and neurasthenia, lays much stress on the psychic origin of the former and the histophysiological basis of the latter. It is quite true that in neurasthenia the evidences of cellular change are more apparent than in hysteria, but in the latter one cannot doubt that some change, probably histophysiological, occurs as well as in neurasthenia. In grave cases of hysteria with pronounced anesthesia, motor paralysis and other striking symptoms, this view is not so likely to be called in question, but it is equally correct when applied to milder cases with hypesthesia and light grades of paresis.

The point is not that histophysiological changes or deficiencies are not present in hysteria, but these are not demonstrable as in neurasthenia, and are more under the control of the will than are neurasthenic phenomena. Even in neurasthenia the will plays no unimportant part, both in the causation of symptoms and as an auxiliary in their relief or cure. I would not, however, have it understood that the part played in the one is at all comparable to that in the other.

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[9] Mettler, L. Harrison. Hysteria and Neurasthenia: Their Nature and Treatment Contrasted. The Illinois Medical Journal, December, 1907.

As has often been repeated, the diagnosis of hysteria from neurasthenia, as from other nervous and mental disorders, is to be made in the first place by the presence in the former of the universally recognized stigmata or landmarks of the disease. These are not present in neurasthenia in which paresthesias take the place of anesthesias, fatigability of paresis, lack of mental concentration of self-absorption, alterations in power of alterations in character. and so on through a list of well-known differences, such as have been indicated by Mettler.

Dana, in his "Partial Passing of Neurasthenia," has given to the profession the best methods of separating this disorder not only from hysteria, but from such mental affections as psychasthenia, hypochondria and melancholia. He has indeed shown that so-called neurasthenia is in many instances only a sort of understudy of some type of insanity, or the threatening stage of some psychosis, which the patient, under wise management, in some instances escapes.

As Babinski and indeed many others have emphasized, when attempting the differentiation of hysteria from neurasthenia, as from organic diseases and from insanities, the somewhat frequent combination of hysteria with these disorders, mental and bodily, must always be kept in mind. That part of the combination which disappears under the influence of persuasion, Babinski would have us understand, is always the part, and the only part, which can be properly classed as hysterical.

In connection with the discussion of hysteria the subject of simulation is one of the utmost importance. In approaching this subject one must have first clearly in his mind the fact that hysteria is not simulation and simulation not hysteria. Hysteria is a functional nervous disease with certain definite phenomena usually classed as stigmata. Good authorities may differ as to the number and character of these manifestations, but all are agreed as to some of them. The subject is clouded by the fact that while hysteria and simulation are not interchangeable terms, the hysteric may, under certain incitements, exaggerate or even simulate. The same temperament which gives rise to true hysteria under sufficient exciting causes seems sometimes to induce feigning. A great difficulty then arises—that of separating the fraud or malingering from the genuine phenomena of a disease—whatever the immediate cause and the nature of this disease may be. Wrong may be done by the lack of careful consideration in such cases.

A desire for sympathy is well known to be present in genuinely hysterical persons, and this fact has been observed by many, and as has been stated by a recent writer, leads to "self-inflicted wounds with consequent sores that will not heal, the vomiting of blood sucked from a tooth and swallowed, and high temperature obtained by friction on the thermometer either in the mouth or rectum, emaciation from supposed starvation and vomiting of all food." (Starr.) To these might be added other manufactured symptoms.

While the existence of such factitious phenomena should put the investigator on his guard, it should not, as some would have us believe, always induce him to regard the whole case as one of fraud. In some instances it is confirmatory rather than antagonistic to the view that the case is fundamentally one of true hysteria. When hysterical convulsions, anesthesias of the classical hysterical type, aphonias, contractures, paralyses, contractions or reversals in the visual field are present in a given case, the existence of affections and conditions which may be assigned to feigning cannot shut out the basic diagnosis of hysteria. Nevertheless cases of pure fraud or malingering should not be classified as hysterical.

Some of the older writers, and careless observers and writers in all periods, have classed as hysterical phenomena clearly due to pure fraud, to insanity or to mistake.

It is scarcely necessary to call attention as instances of pure fraud to cases in which urine or blood is said to have been discharged from parts like the ears, the eyes, the breasts or the navel; to cases in which dismembered spiders or frogs, dead or alive, have been ejected or removed from various bodily receptacles; to cases of ulcerations, eruptions and hemorrhages, so evidently factitious as to need no discussion. Among the examples of fraud which have been reported as instances of hysterical phenomena are some of the extraordinary cases of anuria and ischuria —those which have been recorded as not having passed urine for one or two months, repeated catheterizations revealing an empty bladder. False pregnancy, sometimes considered in connection with phantom tumors, and spoken of as an hysterical phenomenon, is due rather to mistake, fraud or melancholiac obsessions or delusions. Hemorrhages from the stomach, bladder and vagina, while they may occur in the hysterical, are either produced by the patient or are due to causes independent of the hysteria—to bodily injuries, for instance, such as are received in serious accidents. Fever, or rather unusually high temperature, reaching sometimes as high as 110° or 112° F., and reported as hysterical, is also fraudulent, although in a few instances high authorities have reported such cases as genuine. Other examples of simulated, or rather factitious, symptoms regarded as hysterical are dilated or contracted pupils produced by the use of a mydriatic or a myotic.

Recognizing that simulation and hysteria are not identical or interchangeable, the next question is, how is the distinction to be made between hysterical and simulated phenomena? This query is not so easy to answer, as it may at first sight appear. It is easy enough to recognize, as fraudulent and not hysterical, symptoms or conditions which common sense indicates as impossible of occurrence except through fraud. It is when we come to some of the more usual phenomena of hysteria that the greatest difficulties arise—those phenomena for instance which Babinski unhesitatingly places within the hysterical class—convulsions, anesthesias, etc.

Babinski and others following him lay much stress upon the view that all pithiatic, that is hysterical, phenomena can be *exactly* simulated, and in their writings even convey the idea that this simulation is comparatively easy. This way of looking at the matter should not be accepted too readily. To simulate with great precision the serious phenomena which form the hysterical syndrome in some cases is more or less difficult. Every examiner again and again has seen a patient suffering from hysterical anesthesia evidence this by wincing or by speech at the crossing of the median line, and this without suggestion. Even if more or less suggestion is exercised in such an examination the exactness with which the change in the appreciation of sensations takes place in the crossing is beyond the power of simulation. The same might be said about contractions and reversions of the visual field.

Babinski would have us believe that moral considerations above all else should govern us in our diagnosis.

It is true that the incentive to fraud, as in litigation cases, must always be borne in mind, but I would rather say that the separation of hysteria from simulation in cases in which genuine hysterical phenomena are apparently present, is to be made by a consideration of the manner in which the patient does or does not present a symptom picture which, however readily simulated in some of its details, is almost impossible of such simulation in its entirety.

It is much the same with hysteria as with insanity; simulated insanity is almost universally recognized as rare by competent observers and writers, that is the simulation of insanity by one not insane. The form of insanity most frequently simulated is that of comparatively simple type, like mania or dementia, and even here it is difficult to thoroughly carry out the feigning. In those forms of insanity which present a more or less complex or elaborate train or system of phenomena, in the typical forms of paranoia for instance, with delusions of persecution or of self-exaltation, the phenomena being peculiarly grouped, simulation is impossible except to one who has intelligence or training and has closely observed the phenomena of the type the feigning of which is attempted. In grave hysteria likewise, where as a rule, although not without exception, the syndrome is made up of a collection or combination of sensory, motor, visceral and psychic phenomena, simulation is almost equally difficult. It is the inexactitude and incompleteness of the reproduction of the phenomena which should most claim the attention of the diagnostician.

In connection with the discussion of the nature and diagnosis of hysteria, the so-called traumatic neuroses necessarily come into prominence. Usually these are regarded as functional nervous disorders in which the elements of hysteria and of neurasthenia, separate or combined, are present. For the purpose of this discussion neurasthenia may be eliminated, although it plays a part of much importance in many of the cases.

Accepting the theory that all cases of hysteria are due to suggestion, the view has gained ground that not only are most of the traumatic cases due to pure suggestion, but that this suggestion in many instances has had its source in the examination by physicians of those injured or alleged to be injured. The rôle played by direct injury in the production of the nervous disorders presented by litigants by some has been belittled almost to the point of extinction. The mental element in causation, as well as in the clinical picture of the traumatic case, has been given a position of importance so great as practically to obscure all other etiological factors.

It is one thing, however, to recognize the psychic element in hysteria and quite another to believe that physical agencies may play no part in its production. In the majority of accident cases actual physical injury occurs, and the extent and character of this often has a determining influence upon the extent and character of the nervous symptoms presented. To exclude physical injuries entirely from the etiology of the traumatic neuroses would indeed be a happy solution for those who are called upon to compensate for injuries received or alleged to have been received, but this cannot be done with justice to all parties concerned. It may be said that such injuries simply act as suggesters, but even admitting this, merely for the sake of argument, they are the most powerful of suggesting agencies.

In examining cases in which injury is alleged great care should, of course, be taken not to suggest to a patient the presence of special symptoms, but even when this is unconsciously or inadvertently done, the clinical picture revealed by the examination is often such as could not be suggested at a first examination. It is easy to understand that a person may have the idea of anesthesia in this or that part of the body suggested by the use of instruments or appliances which are evidently intended to determine the presence or absence of sensation of touch, or pain or temperature; but it is not easy to comprehend that anesthesia thus determined shall extend exactly to the median line of the body or have a peculiar segmental localization. It is easy to understand that a patient may have suggested to him by methods of examination, even an impairment of vision or a diminution of motor power, or some other single, or perhaps more than one, symptom in the complex of hysteria; but it is not so easy to believe, as I have already indicated, that a train of disturbances, sensory, motor, visual and visceral, can be elicited by a single examination. However these phenomena originate, by examination or by the unaided work of the mind of the one in whom the symptoms appear, even those who hold to the doctrine of suggestion as the active agent in the production of the phenomena are willing to admit their genuineness.

Litigants have their rights and injustice may be done by the too ready acceptance of the doctrine of purely mental or psychic causation in the production of hysteria. Two or three years since an opinion was rendered by the Supreme Court of the State of Pennsylvania which in effect held that damages cannot be recovered for injuries resulting from fright and not from actual physical harm. That fright alone may cause the most distressing result to health, or be even the chief cause of death, in some particular instance, might easily be substantiated by facts which have become historical. In most cases of fright, especially when this comes on in connection with an accident, this psychic phenomenon is itself associated with some serious physical condition and may be directly dependent upon physical cause. Fright assuredly may be an element in those cases in which, as the result of collision or sudden jar or fall, the individual so falls or is so projected as to be bruised or dazed, or otherwise evidently subjected to harm and distress. In these cases, and not improbably in all cases, the central nervous system, and especially certain portions of the brain, are temporarily the subject of important change from their usual normal state. While one may not be able to translate in exact explanatory terms such old and well-worn expressions as nervous shock and cerebral or cerebrospinal concussion, there can be no doubt that some physical perturbation actually occurs before or coincident with the psychic disorder, fright, or whatever else, alleged to be the chief agency in causation.

From the foregoing the following conclusions may be reached:

(1) Hysteria is a disease called functional, because its material pathology is not understood, although it has such pathology.

(2) It is a disease which has for its basis a constitutional condition spoken of with more or less accuracy as temperament, neuropathy or degeneracy.

(3) It is a disease which manifests itself by well-defined symptoms, motor, sensory, vasomotor, visceral and mental.

(4) Hysteria may be caused in a variety of ways, the chief of which is suggestion, although emotion, physical injury or disease and other causes may enter.

(5) Emotional phenomena are frequently present in hysteria.

(6) Hysteria is favorably influenced and sometimes cured by psychotherapy, but may require for its cure auxiliary measures, such as rest, drugs, food, massage, electricity, fresh air and change of scene.

(7) Hysteria is a psychoneurosis, not in a technical sense an insanity, and must be differentiated from psychasthenia and all the accepted forms of insanity.

(8) Hysteria must be differentiated from neurasthenia, although hysteria and neurasthenia are often combined in the same case.

(9) Hysteria is not simulation, although hysteria and simulation may be present in the same case.

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OSTEOPATHY: DOES IT OFFER ANYTHING NEW?

BY N. S. YAWGER, M.D.

> The physician must be experienced in many things, but assuredly also in rubbing. For rubbing can bind a joint that is too loose and loosen a joint that is too rigid. And again, rubbing can bind and loosen, can make flesh and cause parts to waste.—HIPPOCRATES, 380 B. C.

Seldom does one hear of such enthusiasm over one's own work as was expressed by Balfour[1] upon the restoration to health of Madam Ray, a French lady, who had been a great sufferer from diverse gouty and rheumatic manifestations and had not walked a step for eight years. He said: "I congratulate this excellent, meritorious woman upon her restoration to independence; I congratulate those who are still martyrs to rheumatism; I congratulate all mankind, that a cure is at last discovered for one of the most harassing and painful diseases to which human nature is liable, a disease in its nature so obstinate as to have hitherto set at defiance the utmost efforts of the healing art." This cure, he states, he effected by friction, percussion, and compression alone, and he seems to have used these methods successfully in many cases and for various conditions. Further on this physician states: "To the honor of discovering the utility of Compression and Percussion in Rheumatism, to the honor of discovering and introducing into practice the power of Percussion, I have an exclusive claim; and I venture to assert that it will be found a discovery of no ordinary importance."

That osteopathy is but an imitation of foreign methods of treatment is not a new contention. There are, however, certain matters of importance in this connection, which perhaps have not heretofore been brought out.

I wish to state by way of explanation for this article that I am familiar with the teaching of massage and Swedish movements in our American hospitals. I have seen these manipulations given abroad, and have studied the methods of foreigners after the manner of treatments given in the foremost Swedish and German institutions. For some time I have paid special attention to the class of cases in which it is probable that osteopaths get their best results when they get them at all, and have written at some length about these conditions, reporting cases both in this country[2] and abroad.[3]

Chronic rheumatic disturbances are far more frequent than we formerly believed, and great relief may be given in these cases by proper treatment. Rheumatic headaches are very common; infiltrated and tender areas are present in many persons on either side of the spine; lumbago, unilateral sciatica, torticollis, and some so-called cases of neuritis often have a common rheumatic origin.

A not infrequent error we are making is in palpating a painful and tender abdomen and being misled into believing we are dealing with adhesions, appendicitis, floating kidney, gall-stones, and so on. Many normal appendices have been found at operation, and in some of these cases it is the abdominal muscles that are at fault. I am one with Abrams[4] in the belief that pseudovisceral disease is common, and know of a number of mistakes in diagnoses because of these conditions. I am, however, somewhat at variance with him as to the cause of these pains. As I understand it, he considers them to be of neuralgic nature, root pains, and that almost always muscle spasm is demonstrable. Many of these cases I consider rheumatic in origin. Doubtless there is some spasm in the muscles, but there is often something more than this present. I know of suspected cases of appendicitis having cleared up after daily vigorous massage of rigid abdominal areas. I have even seen an acute attack of general muscular rheumatism follow these vigorous treatments where elimination has been imperfect through neglect of keeping the bowels, kidneys, and skin active.

During these treatments, if good elimination is not maintained, patients will ofter complain of headaches, neuralgic and rheumatic pains in various parts of the body. These symptoms are probably due to the setting free of rheumatic matter, whatever this substance may be.

Careful observers have gone so far as to classify these muscular thickenings according to the sensation produced upon palpation into swellings, resistance, and indurations.

In rheumatic subjects the fasciæ and ligaments may also be indurated, and furthermore, deposits may be present in the fat. Surgeons at times observe little nodules in the abdominal fat, some of which are of this variety. Occasionally, by grasping through the skin of the abdomen with the fingers, ridges or nodules may be felt in this adipose layer.

A very interesting case came to my notice recently—that of a woman physician who had suffered from rheumatic headaches for years; of this she was cured through a vigorous course of massage. She then underwent treatment for the reduction of fat over the hips. A few hours after the first vigorous treatment for the breaking up of the fat globules she developed a typical attack of gout in one of her great toes. It appeared suddenly, and the redness and swelling were attended by the most excruciating pain. On the following day the symptoms subsided. She had never experienced such an attack before.

I have observed that a most frequent cause of pain in rheumatic abdominal muscles is gaseous distention of the abdomen. Given then abdominal muscles in this indurated condition, it takes but slight distention to cause stretching of the diseased muscle fibers, thereby producing these obscure pains.

This condition may be demonstrated by the simultaneous presence of pain and tympany, and marked tenderness in the rigid muscle areas, while pressure affords relief. With the subsidence of the tympany the pain ceases, and the rigid areas are much less tender.

Having heard a great deal about the osteopaths, and having read some of their literature, I am reminded of the statement made by a critic after the review of a certain work; he said: "There is a great deal in this book which is *new* and a great deal which is *true;*" then he added: "But it unfortunately so happens that those portions which are *new* are not *true,* and those portions which are *true* are not *new!*"

The osteopaths have become very numerous, and judging from this their patrons must necessarily be plentiful. They claim legal recognition in all States except two, and in these latter are allowed to practice. The business outlook for these workers seems favorable since similar schools have from time to time been founded—Naprapathy, which purports to be a cure through "thrusts" over the ligaments, and Spinal Adjustment, which appears to be an abbreviated and localized form of osteopathy; then there is Chiropractic, and the numbers who have launched forth upon Mechanotherapy are too numerous to mention.

Basically, all of these various systems are the same.

As soon as we shall come to a better knowledge of an important group of cases which can be cured by vigorous massage, movements, and vibrations, and apply these measures properly, the decline of osteopathy shall have begun, as shall the thwarting of the establishment of similar schools making preposterous claims and giving cheap courses of instruction which shall lead to all manner of doctors' degrees.

We have heard much from the followers of Still, of Missouri, about what the osteopaths have discovered, and often their claims for State recognition are based upon this, but upon analysis it appears that their only discovery is in the name osteopathy, and to this they hold undisputed claim.

With a seeming realization of the absurdity of their contentions, that of dislocations as the etiological factor, they shifted to subluxations and muscular contractions, and finally settled upon nerve irritations This latter was taught by the Swedes long ago, and was particularly elaborated by Dr. Kellgren, who located in London; of him more shall be said later.

In osteopathy stress is laid upon manipulations in the region of the third cervical vertebra. The bones there are not out of place, but what they often actually accomplish is the breaking up of adhesions and infiltrations, which are exceedingly common in this region.

Their work may possibly be complete enough to embrace massage, movements, vibrations, and extreme flexions, twistings, and pullings of parts. These possible components of their system have been used by physicians and others abroad for a long time.

Let us consider them briefly:

Massage, it is alleged, was used many hundred years before Christ. It has probably been used in a more or less crude form by most nations. The impetus given it by Dr. Mezger, an Amsterdam physician, led to its more general use throughout Europe. He began his work in 1853, and has unquestionably been the most successful user of this branch of therapeutics. His skill brought to him the royalty from different countries. Physicians came from Germany, Austria, Hungary, and France to study his methods. Among others to give special attention to these treatments were Langenbeck, Billroth, and Esmarch. It was Mezger who classified the manipulations under the four well-known headings of effleurage, petrissage, friction, and tapotement.

A feature of osteopathy is heavy massage. Peter Hendrik Ling, a Swede who began his work during the early part of the last century and later developed his renowned system, used heavy massage and gave special attention to these treatments. Unfortunately, it has been emphatically taught in this country that foreign massage is too vigorous for Americans. This has been a mistake and has given the osteopaths a distinct advantage. In this respect Americans are not different from others, and they can and should be relieved of many rheumatic affections by vigorous treatments.

In the region where osteopaths do most of their work, it is interesting to note that Dowse,[5] England's most enthusiastic advocate of massage, has said: "Let me tell you that ten minutes' work at the spine will increase the volume of the pulse and the temperature generally more than an hour's work at the body as a whole, the spine being omitted."

Movements.—In modern times it was a German named Gutsmuth who began to systematize gymnastic exercise. Then at the beginning of the nineteenth century there appeared persons in different countries intent upon this work, among whom it is sufficient to mention the more prominent; these were "Vater" Jahn, the father of the German Turn system, which, after the Napoleonic wars, had for its object the regeneration of the German nation. In Switzerland the name of Clias stands out prominently, and in Sweden again comes Ling.[6] This indefatigable worker in 1814 secured governmental recognition, which led to the establishment of the Central Royal Gymnastic Institute in Stockholm, and this has ever since been famous throughout the civilized world.

In brief Ling classified movements as *active*, in which the patient performs the movement independently; *passive,* where the operator, grasping the part to be manipulated, performs the movement, the patient remaining inactive; *duplex*, in which the patient performs the movement, the operator offering resistance, or this may be reversed by the patient becoming resistant.

While there are certain standard movements recognized, their possible combinations and modifications are innumerable.

Vibrations.—Again, the mechanical stimulation of nerves was employed to some extent by Ling,[7] but it was not until 1868 that Mr. Hendrik Kellgren, another Swede, developed his method of nerve vibration. This he employed in two ways—by making transverse frictions over the nerve, or by carrying the vibratory movement along the axis of the nerve. Kellgren's brother, Dr. Arvid Kellgren, established a hospital in London where these vibrations were extensively employed, and he has given us elaborate descriptions of this method of manipulation.[8] Vibration by hand has been largely replaced by rapid vibrators whose power is delivered by an electric motor.

Extreme Flexions, Twistings, and Pullings of Parts.—More than twenty years ago, and perhaps much longer, Dr. Sollier was conducting a hospital just outside of Paris, where these severe movements were employed. It has not been possible to find any literature upon this physician's work, and most of my information has come through Americans who were treated there. This institution was well known abroad and patients were attracted to it from different parts of Europe. A Philadelphia woman who underwent this treatment was advised to have the movements continued upon her return home, and for this purpose a Swedish gymnast was selected. Although he is a man of unusual strength and accustomed to vigorous exercise, he informed me that the carrying out of these directions caused him to be greatly fatigued. The exceptional strength and endurance of this particular patient probably accounts for such extreme efforts being necessary, though other persons related a similar but less strenuous experience.

Though massage, movements, and vibrations have quite a wide range of usefulness, it is readily seen that these are not osteopathic findings, since these methods were employed in various parts of the body and for the relief of many ailments, both acute and chronic, long before the founding of Still's school.

In writing upon osteopathy, Dr. J. K. Mitchell[9] sums up the evils of this practice by saying: "The force of the accusation against them lies in their claiming impossible things and in doing harmful ones." Another serious accusation against Still is that of having appropriated the Ling system, and announcing that he discovered osteopathy in 1874; he founded his school in Kirksville, Missouri, in 1892. Still has been charged by one who studied osteopathy with having obtained his information of the Ling system from a tramp masseur, and this is not at all improbable, since for a long time carefully trained Swedish gymnasts have come to this country and settled in our larger cities, and many more have arrived here from the smaller Scandinavian countries. The treatment of disease by manipulations and movements was introduced into New York from Sweden more than half a century ago by two physicians (brothers) named Taylor.[10] I quote from this last reference: "But in that country (Sweden), where these practices are unqualifiedly better understood than anywhere else, where the course of instruction has a duration of three years, and the students must have a knowledge approximate to the degree of B.A. before they enter upon their professional studies, the government does not allow the independent practice of the graduates of its own institution, but requires a certain amount of coöperation between them and the legalized physicians."

Some of our States permit of a wide latitude for osteopathy; they may sign death certificates, and I have recently read of one of their number doing a Cæsarian section.

I do not believe any osteopath is sufficiently well informed to skilfully perform all of the manipulations and movements herein described, but they use some of them, and though they at times get results, they also, through their ignorance of disease, cause injury.

They deny the use of massage and Swedish movements, but though they have adopted these methods, in justice it must be admitted that, to the lay mind, they are demonstrating their usefulness. If the osteopaths have brought forward anything which physicians have not used previously, I should be glad to know it. I am not above learning from any one, but should not like to be in the position of accepting the findings of this so-called new school of medicine, to learn later that all of these treatments have been known for a long time abroad and used there in a much more scientific way.*

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*To those interested in this subject the writings of the following well-known European physicians will be found most instructive: Hartélius, Helleday, Henchen, Norström, Brantings, and Wide in Sweden; Mezger in Holland; Bum, Edinger, Goldscheider, and Leyden in Germany; Kleen in Austria; Vuillemin and Estradere in France; and Balfour, Kellgren, and Dowse in England. There are scores of others.

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THE PHILADELPHIA NEUROLOGICAL SOCIETY

November 27, 1908

The President, Dr. J. W. McConnell, in the Chair

EXOPHTHALMOS ASSOCIATED WITH FACIAL PALSY

By William G. Spiller, M.D.

The history given by the mother of the patient is as follows: G. had a convulsion at the age of three and a half years, and had about five convulsions until she was six years old. The child was healthy at birth and the labor was not difficult nor prolonged. Instruments were not employed. Two children older than the patient are dead, one died from cholera infantum, the other from diphtheria. The patient now 14 years old, had no illness before or at the time the convusions began. The facial palsy and unilateral exophthalmos were first noticed when the child was about five years old. The condition seems to have developed gradually and to have remained unchanged until the present time. There has never been any ear disease. A photograph of the child when she was about two years old represents a normal condition of the face.

The limbs are strong and tactile and pain sensations are normal everywhere. She has not had headache, vertigo, nausea or vomiting. She has complete left facial palsy peripheral in type, *i. e.*, very slight contraction of the orbicularis palpebrarum and no power of contraction in the lower part of the face. The left palpebral fissure is decidedly wider than the right. The faradic current gives prompt response in the muscles on the left side about the mouth and in the chin, but no response in the remaining portion of the facial nerve supply in any strength that can be employed. The electrical irritation of the muscles about the mouth produces a fine tremor in these muscles which persists some minutes after the electrode has been removed. The galvanic current could not be employed at the time of the examination as the machine was not in order.

The left eyeball protrudes somewhat in advance of the right. The pupils are equal. The child stammers, otherwise nothing abnormal is observed in the speech.

The report from Dr. Posey's clinic is as follows: "November 12, 1908. Vision 5/5 in the right eye, and 5/7.5 in the left eye. Left eye is more prominent than its fellow, left palpebral fissure being 3 mm. the wider. Gross ocular movements are good in both eyes and pupils react promptly to light and accomodation stimuli. Neither fundus shows any indication of systemic trouble, and fields of both eyes are normal. There is some refractive error."

Dr. Spiller regarded the exophthalmos as of moderate intensity,[1] and more than could be explained by paralysis of the orbicularis palpebrarum muscle. It had developed with the facial palsy at an early age and during or following a period of convulsions. It was uncertain whether the symptom-complex could be attributed to sinus thrombosis as some other cranial lesions might have been the cause. In the literature he had consulted, exophthalmos is not mentioned as a sign of facial palsy.

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[1] Dr. de Schweinitz kindly measured the exophthalmos February 1, 1909, with Hertel's exophthalmometer. The right eye measured 15 mm., the left 18 mm. Vision of right eye was 6/6; of left eye 6/9.

Extracted from the American Journal of the Medical Sciences, January, 1909

CIRCUMSCRIBED SEROUS SPINAL MENINGITIS.[1]

A LITTLE-RECOGNIZED CONDITION AMENABLE TO SURGICAL TREATMENT.

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A collection of clear fluid in a cyst of the spinal pia-arachnoid is a condition little known in America, as only one case occurring in this country is on record (Spiller, Musser, and Martin, 1903), and only a few are to be found in the German literature. So far, the French and English journals contain no examples. A recent excellent *resume* of the subject by S. Adler makes a complete critical digest by me unnecessary, and yet, because of the little attention paid to the condition, it has seemed desirable to bring the subject before the College.

Almost nothing is known as to the origin of apparently idiopathic collections of fluid within the pia-arachnoid. The fluid may be from the bloodvessels of the pia, as has been suggested, and the mesh-like structure of the leptomeninges may predispose to adhesions, but why these cysts occur in any given case, especially where there are no evidences of inflammation, we do not know. The symptoms are those of circumscribed compression of the spinal cord, and the condition cannot be distinguished clinically from tumor of the cord. It is of great importance, as operation is easy and cure seems to be permanent in typical cases.

In the report of the case of Mendel and Adler, in which an operation was performed by the latter, it is stated that the much-swollen arachnoid protruded through the dura at the operation and appeared violet-blue in color, apparently exactly as in the case we reported. The arachnoid was punctured, and about a teaspoonful and a half of clear fluid escaped in a strong stream and was lost in the bandages. The intra-arachnoidal cavity in which the fluid had been contained was found closed at all parts by adhesions. The improvement after this operation was very marked.

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[1] Read at a meeting of the College of Physicians of Philadelphia, December 2, 1908.

Krause and Oppenheim made their first report of this condition in 1906, but they did not refer to previous work on the subject. This was not extensive, and consisted of the reports of three cases (Schlesinger, Schwartz, and Spiller, Musser, and Martin[2]). Schlesinger, in his well-known monograph on spinal tumors, gave the history of a case in which a cyst of this character was found at necropsy. Schwarz's case (1897) was one with the symptoms of syphilitic spinal meningitis, and after death intense meningomyelitis was found, with a meningeal cyst. This seems to have been a case different from the typical examples, in that closure of a space within the pia-arachnoid might easily occur from the intense meningeal inflammation. Neither of these cases was with operation. The first case reported anywhere with operation was the case by Spiller, Musser, and Martin.

In that case a delicate pial cyst was found and evacuated by Dr. Edward Martin. The operation was on June 30, 1902. The patient had suffered intensely. She has been reëxamined by Dr. Musser and me within the past few weeks, and she has now no symptoms of her former trouble except slight pain in one buttock when she presses over the scar made by the operation. She has gained much in weight and is in excellent health.

This is not only the first case with operation, but is the only one on record in which recovery has persisted after operation so long as six years and five months.

Krause, in operating on 22 cases of spinal compression, expecting to find tumor, found circumscribed serous meningitis in six, although it is not certain that this was the only lesion. Adler remarks that the condition is not a scientific curiosity, as some may think, but is of practical significance. He refers to a case seen by Stroebe. A very similar condition on the under surface of one cerebellar lobe has been described by Placzek and Krause.

The associated lesions that have been found are: Necrotic osteitis of the vertebræ with pachymeningitis (Krause); scar-like adhesions between the dura and pia, probably with spinal gliosis (Krause); bony projection on the inner surface of a vertebra (Krause); and meningomyelitis (Schlesinger, Schwarz).

As Adler remarks, no cause was found in the case reported by him with Mendel, nor in one case of Krause, nor in the case of Bruns, nor in our case. Adler is inclined to believe that in the case reported by himself and Mendel tuberculosis may have been present, as the patient had had pulmonary tuberculosis; and he hesitates, on the basis of four cases, to speak of an idiopathic form of circumscribed serous meningitis. He suggests the possibility of spinal puncture, and refers to the fact that after operation the fluid does not seem to collect again. Bruns, stimulated by Mendel and Adler's[3] paper, publishes his case more in detail, and reports almost complete recovery two years after the operation.

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[2] Univ. Penna. Med. Bull., March and April, 1903.

A case reported by W. C. Krauss hardly belongs to this group of cases, as the cyst was not confined to the pia-arachnoid. The diagnosis of spinal tumor was made, but an extradural cyst in eroded vertebræ was found. Dr. Roswell Park states, however: "It seemed evident that there was a cavity with eroded walls connecting with the spinal canal, containing mostly fluid, with a little semi-solid tissue material."

It is questionable whether the spinal cyst described by Schmidt should be included under this type. It was intradural, but not a cyst of the pia. Its wall had the consistency of the dura.

Although the literature on circumscribed serous spinal meningitis, as shown by this *resume*, is meagre, it is sufficient to stimulate us to pay attention to the condition and to encourage us in the belief that certain cases with the symptoms of spinal tumor may be more amenable to surgical intervention than are many tumors.

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[3] Berl. klin. Woch., August 31, 1908. The literature is given in this paper; Bruns, ibid., September 28, 1908; Krauss, Brain, 1907, xxx, 533; Schmidt, Deut. Zeitsch. f. Nervenheilkunde, 1904, xxvi, 318

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THE SYPHILITIC FORM OF MULTIPLE SCLEROSIS.

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AND

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Syphilis may bear a resemblance to multiple sclerosis both in its symptomatology and in its pathology. One of us, in association with Dr. Camp,[1] has reported a case which clinically appeared as one of multiple sclerosis, from the symptoms of ataxia, intention tremor, scanning speech, pallor of the temporal side of one optic nerve, and vertigo. Those who saw the case regarded it as one of multiple sclerosis, and yet the microscopic examination showed round-cell infiltration and thickened vessels without sclerotic patches—the common findings of syphilis of the nervous system.

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[1] American Journal of the Medical Sciences, June, 1907.

We propose in this paper to consider the pathological aspect of the subject, and to show that syphilis may cause sclerotic patches in the central nervous system, resembling the degeneration of multiple sclerosis without necessarily producing the symptoms of the latter disease.

S. D., No. 445, was admitted to the Philadelphia General Hospital, in the service of Dr. Mills, November 28, 1907, and afterward came into the service of one of us (Dr. Spiller). She died March 17, 1908. She entered the hospital because of pain and weakness in the lower limbs and inability to walk. She had had three children and four miscarriages, and denied syphilitic infection. When she entered the hospital she stated that five weeks previously she had first noticed the weakness of the lower limbs, and about five days later she began to have severe pain in these limbs below the knees. She had lost control of the bladder and bowels. Her age was given as fifty two years.

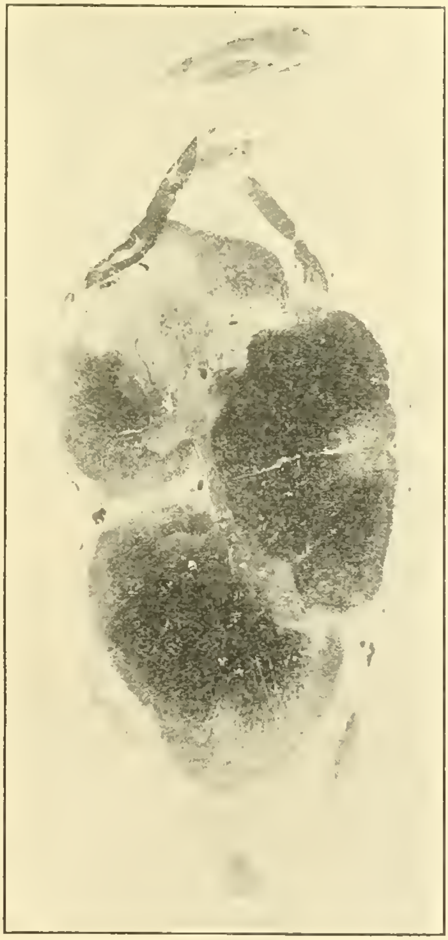
The pupils were equal and regular. The iridic reaction to light, accommodation, and convergence was preserved. The grip of each hand was somewhat diminished, as was also the voluntary power of the upper limbs. The biceps and triceps reflexes were exaggerated.

The lower limbs were edematous, and were kept flexed at the knees, and movement of these limbs caused much pain. The patellar reflexes could not be obtained, probably because of the position of the limbs. There was a suggestion of ankle clonus on the left side.

An examination made by Dr. McConnell, in Dr. Mills' service, December 1, showed atrophy of the right interosseous muscles. There was no tremor of the limbs. The patient had very little voluntary movement of the lower limbs. Sensation seemed to be normal in all forms.

She remained in much the same condition until her death, March 17, 1908.

The symptom-complex was therefore one of spastic paraplegia of the lower limbs, with contracture of the limbs and pain produced by passive movement of these limbs, probably because of the contractures, exaggeration of tendon reflexes in both upper and lower limbs, although the reflexes could not be well demonstrated in the latter because of the position of these limbs, and loss of control of the bladder and rectum, with preservation of objective sensation. The typical symptoms of multiple sclerosis were absent, as there is no



Photograph showing sclerotic plaques.

mention of scanning speech, intention tremor, nystagmus, etc., and the diagnosis of multiple sclerosis was not made.

Numerous areas of sclerosis are found throughout the cervical and upper thoracic regions of the cord, and are confined to these regions. Some of these resemble very closely the degenerated areas found in multiple sclerosis. They affect the gray as well as the white matter, are of various shapes and sizes, and in general are not sharply defined from the surrounding normal tissue, but shade off gradually into the normal tissue. There are, however, regions in which the definition between the degenerated and normal tissue is quite sharp, as in multiple sclerosis.

The small vessels of the cord are much thickened in many regions; and the neuroglia around many of them is denser than in other places, making it appear as though a perivascular sclerosis were the origin of some of the sclerotic patches.

Many of the vessels in sclerotic foci are distinctly thickened but there cannot be said to be any increase in the number of the vessels in these foci.

Round-cell infiltration is distinct in the pia, but is of only moderate intensity. The vessels of the pia are somewhat thickened, although they do not show a very distinct proliferation of the intima.

Some of the nerve cells of the anterior horns show intense pigmentation, and in some places the nuclei are displaced to the periphery of the cells.

Secondary degeneration is not very distinct.

Round-cell infiltration of the pia is present in the optic nerves. It is not intense anywhere.

While the resemblance between the sclerotic areas in this case and those of ordinary multiple sclerosis is on superficial examination very striking, the two conditions differ in essential features. The round-cell infiltration of the pia; the gradual shading off of the sclerotic areas into the normal tissue instead of the sharp line of differentiation; the vascular and perivascular sclerosis in regions of the cord where there is no distinct focus of sclerosis, indicate that the process is syphilitic, and distinguish it from the disease known as multiple sclerosis. And yet it is well to bear in mind that in multiple sclerosis the definition between the sclerotic and normal regions is not invariably sharp, and that we may have thickened bloodvessels within the sclerotic foci.

Von Bechterew, in 1896, spoke of the syphilitic disseminated sclerosis, in which areas of sclerosis occur in the brain and spinal cord, as a little known type of syphilis. In the thirteen years that have elapsed since the appearance of his paper few important contributions with necropsy reports have been made to this subject.

Von Bechterew discussed the question as to whether the vascular thickening in the sclerotic areas was primary and the cause of the surrounding sclerosis, or was secondary to this sclerosis. The syphilitic nature of his case was shown by a gumma of the left optic thalamus and the characteristic disease of the basilar artery. The foci of sclerosis were found about much thickened bloodvessels. Thickened vessels were observed in parts of the brain without the surrounding sclerosis, and were present in all the sclerotic areas, and von Bechterew has no hesitation in saying that the thickening of the bloodvessels was primary. He found secondary degeneration in the spinal cord and brain as a result of the sclerotic foci, also hemorrhages in and near the foci, and softening about some of the foci, all of which lesions are not characteristic of multiple sclerosis.

Clinically, von Bechterew's case was not typical of multiple sclerosis. The nervous symptoms appeared one year after the syphilitic infection. After study of the limited literature on the subject he was forced to conclude that previous to the appearance of his own paper the disseminated syphilitic sclerosis was not well established.

He attempts to differentiate clinically between the true and the false multiple sclerosis. In his case weakness of the lower limbs was an early symptom; this he thinks uncommon as an early sign of multiple sclerosis, and in this disease it never is a pronounced paraplegia. One may with reason doubt the correctness of this statement. Disturbance of the bladder was an early symptom in his case; this is seldom an early symptom in multiple sclerosis. The early paresthesia of his case likewise is uncommon in multiple sclerosis. Local tenderness of the vertebræ to percussion, present in his case, he regards as common in syphilitic spinal affections, but it has not been observed in multiple sclerosis. The tremor of the limbs and head, the scanning speech, and nystagmus were not present in his case, and he thinks it important that attention should be paid to these symptoms, as further observation may show that they are usually or always absent in the syphilitic disseminated sclerosis. The case of Spiller and Camp shows, however, that intention tremor and scanning speech may be caused by syphilis even without the formation of plaques. The syphilitic form seems to have a more acute course; in von Bechterew's case the duration of the process was one year and three months, and yet acute multiple sclerosis is recognized. The syphilitic form von Bechterew mentions is influenced by antisyphilitic treatment, but this seems to us most unreliable in distinguishing the disease from multiple sclerosis, as changes in the intensity of the latter disorder are common. The early paresis of ocular muscles in his case he also employs as diagnostic.

In von Bechterew's[1] case the foci were in the brain as well as in the spinal cord; in our case they were only in the cord, and yet clinically and pathologically the two cases have much in common.

Catola has given an excellent *resume* of the views of various authors on syphilis as a cause of multiple sclerosis as held at the present time. Some writers have attempted to distinguish a form of sclerosis with somewhat indefinite boundaries, complete degeneration of medullary sheaths and of axis cylinders, and secondary degeneration, as distinct from the usual type, and as having syphilis as one of its causes (Marie).

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[1] Archiv f. Psychiatrie, 1896, xxviii, 742.

Müller regards the syphilitic multiple sclerosis as secondary to arteritis.

In the opinion of some, the syphilitic multiple sclerosis is to be distinguished by degenerative changes of the nerve cells and axis cylinders, by secondary degeneration, and by thickening of the vessels within the sclerotic areas.

There is such a difference of opinion as regards the tissue first affected in multiple sclerosis that we have no means of reconciling the conflicting views; it is uncertain whether the vessels are first affected or the neuroglia, and in many instances thickened vessels have been found in the sclerotic patches. By some multiple sclerosis is considered a congenital proliferation of neuroglia.

The conclusions reached by Catola are that the distinctions between the different forms of multiple sclerosis are unreliable, and that syphilis cannot be eliminated as a cause.

In the first case reported by him the sensory symptoms were more pronounced than is customary in the true multiple sclerosis. The patient had pains in the back, shooting pains and diminution of sensation in the lower limbs, and loss of stereognostic perception. The findings were: Thickening, of the pia over the base of the brain and over the spinal cord, with infiltration by lymphocytes and polynuclear cells, sclerotic plaques in the brain and cord, sclerosis of all the vessels of the pia and spinal cord, with some perivascular cellular infiltration. Secondary degeneration was absent. Catola[1] thinks the syphilitic nature of these lesions is shown by the perivascular alteration. From the illustrations of this case many of the sclerotic areas appear to shade off gradually into the normal tissue.

In his second case sclerotic areas were found and the vessels of the cord were thickened, but perivascular round-cell infiltration and round-cell infiltration of the pia seem to have been inconspicuous. It is stated that the perivascular spaces contained occasionally some round cells. Secondary degeneration was absent. In the summary of the case the vascular lesions are described as thickening and degeneration of the walls of the vessels. The author might well have taken more space to establish the syphilitic nature of these sclerotic plaques, as beyond a syphilitic infection the syphilitic character of the sclerotic areas is at least open to question.

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[1] Nouvelle Iconographie de la Salpêtrière, 1906, xix, 337.

Nonne,[1] in the second edition of his work on syphilis, having the date of 1909, says that multiple sclerosis in its typical symptom complex is not caused by syphilis. The course of the syphilitic form is different and the typical symptoms of multiple sclerosis—scanning speech, rotatory nystagmus, intention tremor, and the general tremor of trunk and head—are not observed in cerebrospinal syphilis. Syphilis in his opinion certainly plays no part in multiple sclerosis. Schuster's clinical case he does not accept, but he acknowledges that the "formes frustes" may be difficult to distinguish clinically from cerebrospinal syphilis.

Certain authors (Orlowsky,[2] Thomas and Long[3]) have held that syphilis and multiple sclerosis have occurred in the same person. The possibility of the simultaneous existence of these two diseases in one nervous system must be acknowledged, but it is not a probability. The case of Thomas and Long is one in point, and called forth a vigorous protest from Ladame,[4] who regarded it simply as an unusual example of spinal syphilis.

Oppenheim[5] expresses himself very forcibly, and in his fifth edition of his text book has not seen fit to alter his opinion that syphilis has nothing to do with multiple sclerosis, although syphilis may cause multiple foci of degeneration, as demonstrated by von Bechterew, but he states that Catola has made it probable that syphilis may cause a disease related to multiple sclerosis. Oppenheim's study of syphilis of the nervous system has been so extensive that his opinion demands respect.

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[1] Syphilis und Nervensystem, 1909.

[2] Neurologisches Centralblatt, 1897.

[3] Comptes rendus de la Soc. de Biologie, 1899.

[4] Revue Neurologique, 1900, viii, 66.

[5] Lehrbuch der Nervenkrankheiten, fifth edition, 1908.

Williamson,[1] in his recent work, states that "The disease has no relation to syphilis," calling attention, however, in describing certain anomalous syphilitic lesions, to the occurrence of cases resembling disseminated sclerosis in the evidence of multiplicity of lesions.

We might refer to the views of Gowers and other writers expressing doubt as to the relation of syphilis to multiple sclerosis, but with one or two exceptions we have confined our quotations to recent authors who have expressed an opinion on this subject.

The conclusions we have reached from our investigation of the case reported in this paper and other cases are:

1. Syphilis may in rare instances cause a symptomatology indistinguishable from that of typical multiple sclerosis, and this without the formation of sclerotic plaques, but by the ordinary lesions of syphilis, viz., arteritis and meningitis.

2. Syphilis may produce sclerotic plaques in the spinal cord resembling those of multiple sclerosis, without producing the typical symptoms of this disease. These plaques have not the sharp definition seen in most cases of typical multiple sclerosis, and yet multiple sclerosis may exist without plaques sharply defined from the normal tissue. The syphilitic form of multiple sclerosis presents round-cell infiltration of the pia and thickening of the bloodvessels. Thickened vessels with a small amount of perivascular sclerosis will usually be found in certain regions without the formation of plaques, though plaques may be present at other levels of the cord. In some places slight neurogliar proliferation without thickened vessels may be detected, resem-

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[1] Diseases of the Spinal Cord, 1908.

bling in no way true plaque formation, but appearing more in the nature of slight diffuse secondary degeneration. Gumma also may occur in the brain. Secondary degeneration is more common in the syphilitic type, but does occur rarely in multiple sclerosis. Nerve fibers are not always completely degenerated in the syphilitic plaques. Careful and thorough examination will almost always, if not always, make a diagnosis possible between the lesions of syphilis and those of multiple sclerosis, even though they may have a decided superficial resemblance.

We are indebted to Dr. Alfred Reginald Allen for the photograph.

SPECIAL FEATURES IN THE SYMPTOMATOLOGY AND PATHOLOGY OF ANEMIA OF THE CENTRAL NERVOUS SYSTEM

BY

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SPECIAL FEATURES IN THE SYMPTOMATOLOGY AND PATHOLOGY OF ANEMIA OF THE CENTRAL NERVOUS SYSTEM.*

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PHILADELPHIA.

AFTER the excellent contributions of Minnich, Lichtheim, and Nonne in Germany, Putnam, Taylor, Burr, Lloyd, Dana, Spiller, and others in this country, Russell, Batten, and Collier in England, and Bastianelli in Italy, there seems little that can be added to the clinical and pathological picture in anemia of the spinal cord. For the most part these observers have developed the clinical and pathological changes incident to this disease. A few have endeavored to establish a distinct clinical entity, while others have concerned themselves with the disease in relation to the pseudosystemic diseases. Moreover, the rôle played by the anemia, the influence of special toxic agents, as well as the part played by the blood-vessels, have been thoroughly discussed in the last decade.

The general clinical symptoms of paresthesias, weakness, ataxia, and spasticity are too well known for me to enter into a discussion of them. The spinal changes consist in symmetrical areas of sclerosis, involving most frequently the posterior and

*From the Department of Neuropathology of the University of Pennsylvania.

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lateral tracts. The lower cervical and upper thoracic segments show the most extensive degeneration. The blood-vessels are congested, and their walls sometimes show hyaline degeneration. The perivascular spaces contain leucocytes and granular material. Besides the neuroglia proliferation, rarefaction of the fibers and may spaces are noted. Where they have disappeared sometimes small hemorrhages are seen. The cells in the gray matter show usually pigmentation.

Certain clinical features are worthy of mention in our series, six of which were studied clinically and pathologically and two only clinically. The onset of this disease is usually gradual, occurring over many months. In Case VI the peculiar feature of sudden onset was noted, the clinical symptoms of weakness and paresthesia developed in twenty-four hours. In Case I we noted the presence of a sharp, almost girdle-like zone of anesthesia, reaching to the umbilicus. This symptom, according to Nonne, is uncommon. It may complicate the diagnosis as it did in one of Nonne's cases, in which a diagnosis of tumor was made. Sometimes the character of the pain is puzzling and Dr. Spiller tells me of a case in which the pain was located in the back at the level of the third thoracic vertebra, and radiated from this situation. The diagnosis of tumor of the spinal cord and anemia, the patient having 3,000,000 red blood corpuscles, was difficult.

Another striking feature in the study of these cases has been the variable character of the symptoms in the different cases. This variableness is also to be noted in the cord changes. Sometimes only the posterior columns are involved. This was observed by Lloyd in the case which he published, and the microscopical sections of which I have again studied. Sometimes the process is seen in both the posterior and lateral tracts; again it may be noted as a diffuse sclerosis of the entire cord. Thus it is quite apparent why these cases may be diagnosed as tabes, disseminated sclerosis, myelitis, and tumor. Nonne has had the opportunity of studying some of the earlier forms of the disease and reports isolated foci of sclerosis affecting various levels of the cord. These apparently have no special predilection for any one system of fibers. From these findings, together with the varying character of the clinical symptoms, in a recent contribution he concludes that this disease is not systemic but focal in character. I fully share this recent view of Nonne, as the material at my disposal (six cases) fully justifies his conclusions. It is interesting to note in passing that he formerly believed in the systemic character of this process, and that only in his last contribution to this subject he returns to the theory advanced some years ago by Leyden and others.

Sometimes, as Spiller remarks, the paresthesia and ataxia may persist for many years with little change in the cord; and, on the other hand, the intensity of the alterations may be much greater than the clinical symptoms suggest. This peculiarity has also been noted by most observers in the character and relation of the anemia to the disease process.

Two unpublished cases, one of Dr. Spiller, the other of Dr. Mills, illustrate this. In Dr. Spiller's case, S. P., female, the physical examination showed a spastic gait, ataxia, ankle clonus on the right side, Babinski's reflex distinct on each side. Sensation for touch and pain was diminished in the lower limbs, almost normal in the upper limbs. There was no involvement of the cranial nerves. The blood count showed 8,160 white blood corpuscles and 5,090,000 red blood corpuscles.

Dr. Mills' case, M. P., was a female, aged thirty-two, with a history of pain in the back and legs but with no physical symptoms except some exaggeration of the knee jerks; no disturbances of sensation, no ataxia or spacity; the blood count showed as low as 2,240,000 red blood cells, 46 per cent. of hemoglobin, and 16,000 white blood corpuscles.

While not definitely proved, the theory of a toxic agent associated with the anemic state seems to be most plausible. In this connection it is interesting to note the incidence of tuberculosis. In two out of six of our cases which came to necropsy, tuberculous lesions were found in the viscera. In a review of the cases reported I find about the proportion of one to seven.

To what extent is the brain involved? In our series of cases we note the presence of convergent strabismus, right facial palsy, inequality of the pupils, and slow response to light. Russell also reports unilateral facial paralysis, and in another case paresis and ataxia of the larynx. Bulbar symptoms have been reported by Putnam, Taylor, and others, while Weisenburg and McConnell have reported a case with double optic neuritis. This is interesting, for the question has arisen whether anemia could produce optic neuritis, some holding that it only produced optic atrophy.

The mental state has been variously pictured, most observers stating that the patient is dull and inattentive. They frequently state that physical examination is unsatisfactory, owing to the dullness of the patient. Nonne reports clouding of the mental state from somnolency to delirium. The majority of my cases showed apathy; two cases were reported as emotional and one as hysterical. Toward the end of the disease in some of the cases delirium and confusion occurred, and in others stupor, but it is questionable if these later states played a part in the anemic picture, owing to the association of a terminal infection. An interesting side light is thrown upon this subject by the clinical studies of Pickett, Grawitz, and Siemerling in various types of anemia. Pickett reported five cases in which the composite picture showed shallow confusion with impairment of the ideas of time and place, with fabrication and relating imaginary experiences, not unlike Korsakow's syndrome. Illusions as well as hallucinations and persecutory delusions were common. Grawitz emphasized the apathy, unfeelingness, and melancholia in some, while in others there were stages varying from excitement and delirium to mania. Siemerling most recently has endeavored to establish a definite clinical picture in this condition and compares it to the state arising in the course of infections and intoxications.

Our cases show that the spinal cord symptoms predominate over the cerebral, and no definite mental syndrome is seen. A review of the literature corroborates these findings, though one must not overlook the fact that this phase of the subject may not have been given sufficient attention.

From the pathological studies of the brain in anemia, the changes found in the brain might account for the train of symptoms described by Pickett and others. Recent experimental studies of Orr and Rows have shown swelling of the perivascular lymph spaces, loss of contour of the cells, chromatolysis, and congestion of the vessels. In my studies of the cortex a similar picture was noted. The pathological changes found in the spinal cord in most of our cases correspond to the usual findings. In Case III, however, we found the unusual feature of involvement of only the left crossed pyramidal tract, the posterior columns were symmetrically and widely degenerated indicating that the process was of some duration. The extent of the lesions reached in two of our cases to the medulla, and one of these could be traced by the Weigert method to its upper border. The involvement included the pyramidal and direct cerebellar tracts. Mott, Russell, Spiller, and others have found the destructive lesions as high as the mid-region of the pons, and Mott and Russell have been able to trace the degeneration by the Marchi method as far as the cortex.

Case I.—H. S., in the service of Dr. Spiller, male, aged fifty-five, had no venereal history; used alcohol moderately. The family and previous medical history was negative. About two years before admission he noticed a creepy sensation and feeling of pain in the right leg, and shortly after the left leg became affected. One year later his gait became unsteady and his feet felt numb. Four months later he could not use his lower limbs and a few months before admission he began to have dribbling after urination.

Physical examination: He could rotate inward the lower limbs very slightly at the thigh; otherwise the limbs were paralyzed. He could not even move his toes. Patellar tendon reflex was entirely absent, even on reinforcement on right side; very faint on left side. Achilles tendon reflex was nearly normal on each side; Babinski's on both sides. Plantar reflex was greatly exaggerated on each side, especially the left. Irritation of the left sole caused the left knee to be flexed three or four times, and contractions occurred in most of the muscles in the lower limb. The cremasteric reflex was preserved on each side. Sensation for touch, pain, and temperature was entirely lost in lower limbs. Sensation for touch, pain, and temperature was diminished over front of trunk below the nipple line. Sensation was lost in irregular areas over the thighs. The movements of the upper limbs were free; the grasp of each hand was normal.

Examination three weeks later showed complete motor paralysis in lower extremities. Sensation was lost to a point at the level of the umbilicus. A band of hyperesthesia was noted above this extending three inches in width. The patellar tendon reflexes were absent; Babinski's reflex present. The grasp was weaker and patient complained of numbness in hands.

Blood examination revealed 11,000 white blood corpuscles, 45 per cent. hemoglobin, 2,580,000 red blood corpuscles.

Microscopical examination: The changes in the spinal cord showed extensive and symmetrical sclerosis of the posterior and lateral tracts from the lumbar to the cervical region. In the lumbar segments the sclerosis was "V" shaped in the posterior columns. The periphery and a portion of the root entrance zone were free. The crossed pyramidal tracts were involved on both sides; only the right direct pyramidal tract was involved. In the thoracic and cervical segments the sclerosis was more extensive, but similar in distribution.

Clinical summary: There was no history of syphilis. The family and previous medical history was negative. The illness began two years before admission with paresthesia and weakness in lower extremities. There was loss of bladder and rectal control. The patellar reflexes were absent; Babinski's present. Motor paralysis was complete in the lower extremities. Sensation was lost to the level of the umbilicus. There was hyperesthesia in a band three inches in width above this area. A diagnosis of myelitis was made.

Pathological diagnosis: Combined sclerosis from anemia. The changes in the spinal cord were symmetrical and involved the posterior and lateral tracts from the lumbar to the cervical region.

Case II.—I. H., in the service of Dr. Mills, aged sixty-eight, male, colored, had a negative family and previous medical history. The present illness began two years before admission with a feeling of numbness and tingling in the legs. This trouble increased with weakness until he was unable to work. About the same time his hands and arms became weak and numb. For the past three months he had had shooting pains in the legs.

Physical examination: The pupils were equal, reacted to light and accommodation. He performed all the movements of the eyes except convergence; attempts at convergence brought inward the right eye at times, the left remaining in the median line. There was no paralysis of the fifth, seventh, or other cranial nerves. In the upper extremities the movements were equally weak on both sides. The limbs were somewhat spastic. Ataxia was shown by the finger-to-nose test. There was very marked atrophy of the shoulder and arm muscles. These muscles were the seat of fibrillary tremors. There was some wasting of the ulna and interosseus groups. The biceps and triceps reflexes present but not increased

Lower extremities: The limbs were weak and ataxia was marked in both legs. The patellar tendon reflexes were increased on the right side and preserved on the left. Ankle clonus was absent. The muscles of the left leg and thigh were more atrophied than on the right. Fibrillary tremors were noted in the atrophied muscles.

Sensation: Response to touch, pain, and temperature were correct in all parts of the face, trunk, and upper extremities. In the lower extremities there was complete loss of tactile sensation in all portions of the thigh, legs, and feet. Hypalgesic areas were found in various portions of the thighs and legs. Thermic sense was undisturbed. Sense of position seemed completely lost in the left and greatly impaired in the right. The mental condition was apparently fair until about ten days before death when there were signs of mental confusion. Just before death he had convulsions.

Necropsy protocol: Catarrhal pneumonia with cheesy encapsulated foci of tuberculosis; brown atrophy of heart; infection of the right kidney and ureter.

Microscopical report of spinal cord: In the lumbar region the areas of degeneration were symmetrical in the posterior and lateral columns. A fair number of fibers in the periphery and continguous to the septum had escaped in the posterior columns, also the root entrance and cornu comissural zones were intact. The crossed pyramidal tracts were symmetrically involved. The direct pyramidal tracts were not involved. In the thoracic region the involvement was more extensive and still showed its symmetrical arrangement. In the cervical region the process was most extensive; only the root entrance zone and cornu commissural zones were free, and here many rarefied fibers were noted. The lateral columns were extensively degenerated. The sclerosis involved the crossed pyramidal, direct cerebellar, and Gowers' tracts. The direct pyramidal tracts were only slightly involved. The pathological changes were those of anemia. Small hemorrhages and slight round cell infiltration of the perivascular spaces were noted. Many new capillaries were seen throughout the sclerosed areas. The cells in the anterior horns showed considerable pigmentation. The medulla and pons showed no changes. Section taken from the cerebral cortex showed marked distension and congestion of the vessels in the meninges and throughout the interior. Their perivascular spaces contained considerable granular material. The ganglion cells were in some instances swollen; chromatolysis was noted, and the nuclei were misplaced.

Clinical summary: Male, aged sixty-eight, had loss of power and paresthesia in lower limbs, extending over two years; ataxia; spasticity in all four limbs. Increased patellar reflex on right side, Babinski's positive. Tactile sensation was lost in lower limbs. There were marked muscular atrophy in upper limbs, and mental confusion nine days before death.

Microscopical diagnosis: Combined sclerosis. Sclerosis was symmetrical and extensive, involving the posterior and lateral columns. In the cortex the chief changes were congestion, cloudy swelling, and chromatolysis.

Case III.—J. W., in the service of Dr. Dercum, aged sixty-one, teamster, admitted specific venereal infection. The clinical examination of this patient was very meager. The present illness began two months previously with weakness and loss of control of lower limbs. Examination showed ataxia, no muscular atrophy, no disturbances of sensation; right facial weakness. The mental state was dull, and at times the patient was delirious and talkative.

Death occurred three weeks later. The clinical diagnosis was specific myelitis.

Pathological report: In the lumbar region no degeneration was noted. In the thoracic segments the posterior columns were symmetrically involved; the crossed pyramidal tract was involved only on the left side, a few of the fibers of the posterolateral tract were also involved. In the cervical segments the sclerosis was most extensive, the posterior columns were symmetrically involved; the crossed pyramidal tract and a few fibers of the posterolateral tract were involved only on the left side. The medulla at the decussation and above showed no involvement of the pyramidal tract, but showed sclerosis in the lateral tract. The pathological changes present showed no evidence of specific inflammation, but the presence of the areas of sclerosis, the vacuolated areas (*Lueckenfelder*), and vascular changes resembled anemia of the spinal cord. Pathological diagnosis: Combined sclerosis.

CASE IV.—J. C., in the service of Drs. Mills and Spiller, aged sixty-two, iron worker, denied syphilis. The family and previous medical history was negative. Present illness: Two years previous to his admission he had phlebitis in both legs and was ill for three months. One year later he became weak in the lower limbs. Following a fall he was unable to move his left leg and soon after lost the power in the right leg. Three weeks later he regained enough power in his legs to move about with the aid of a cane. At time of admission he was unable to walk, and complained of numbness in the hands and legs.

Physical examination: Pupils reacted to light and convergence. There was no weakness of facial muscles, nor muscular disturbance. The tongue was protruded in the median line. No muscular atrophy was noted in the upper extremities. The biceps and triceps tendon reflexes were prompt on each side. The sensation to touch and pain was normal. In the lower extremities the left knee was enlarged and a little warmer to palpation than the surrounding parts. The left leg was partially flexed on the thigh and there was resistance to passive movement. Muscular atrophy was not pronounced. Voluntary movement of the toes and ankles was about normal. The patellar tendon reflex on the left side was exaggerated, while the right was only slightly so. There was no ankle clonus. The Achilles tendon reflex was diminished on each side: Babinski's positive on each side. Tactile sensation seemed to be entirely lost in both lower limbs below the knee and was markedly impaired in the right thigh and about normal in the left. The pain sense was apparently lost in the same areas as the tactile sensation; it was diminished in the right thigh and nearly normal in the left. There was no impairment of sensation to touch or pain over the abdomen or the lower part of the back. The patient had a feeling of constriction passing directly through the umbilicus. There was decided rigidity in the lower limbs to passive motion.

Diagnosis: Lesion of the spinal cord in the lower thoracic region.

Necropsy protocol: The lungs showed tuberculous deposits at both apices. There was chronic interstitial nephritis and also present a large amount of yellowish fat which suggested pernicious anemia.

Microscopical examination: In the lumbar segment the posterior columns were symmetrically involved, but not so extensively as in the thoracic or cervical areas. A triangular area and oval area around the septum were involved as well as the root entrance zone and cornu commissural zone. The direct pyramidal tracts were not involved; the crossed pyramidal tracts were involved on both sides, but more so on the right. The lesions were those of sclerous and vacuolated areas, together with perivascular infiltration and new formation of capillaries. The thoracic segments showed a symmetrical involvement of the posterior and lateral columns more extensive than in the lumbar, but less than in the cervical. Gôwers' tract and the anterolateral ground bundle showed little sclerosis but showed the vacuolated areas. The direct pyramidal tract was involved only on the right side. In the cervical segments the lesions were most extensive. The columns of Goll were entirely destroyed except for a peripheral zone. The columns of Burdach were less involved and the root entrance zone and cornu commissural zone were preserved. The scleroses, vacuolation, and perivascular infiltration showed the same characteristics as in the other segments.

Summary: Male, aged sixty-two, developed illness during a period of two years. The illness began with weakness and paresthesia in the lower limbs and girdle sense. Improvement was followed by remission; weakness and spasticity, increased patellar reflexes, Achilles reflex diminished no ankle clonus, Babinski's reflex was positive on both sides. Tactile and pain sensation was lost in both lower limbs in the same areas; there was no disturbance of cranial nerves.

Macroscopic examination showed tuberculosis of the lungs and kidneys and appearance of anemia in the other organs.

Microscopical examination: Symmetrical and extensive sclerosis and vacuolation most marked in the posterior columns and lateral columns of the cervical and thoracic segments. Direct pyramidal tract was not involved on the lumbar segment. Crossed pyramidal tracts were more involved on the right side. Changes were those of anemia. Microscopical diagnosis was combined sclerosis.

Case V.—M. S., female, in the service of Dr. Spiller, had no specific history, but was an alcoholic three years before. Family and previous medical history was negative. The present illness began after standing for the entire morning. She noticed that her legs were weak and she was able to walk only with support. She complained of numbness and tingling in the limbs.

Physical examination: The pupils reacted to light and accommodation; the former was not so prompt The extraocular movements were normal as was sensation in the face. She drew up the corners of the mouth well on each side, closed her eyes and wrinkled the forehead well. The tongue was protruded in the median line. Upper extremities: The grasp of the hands was poor. The upper limbs were not wasted. Biceps tendon reflexes were exaggerated on each side. There were no sensory disturbances. Voluntary movements and resistance to passive motion were normal. Lower extremities: There was marked paralysis in the lower limbs. She could flex the thigh slightly at the hip and knee. Resistance to passive movements was feeble. Patellar tendon reflexes were markedly exaggerated on each side. Patellar clonus was persistent on each side; ankle clonus on each side. Babinski's reflex was not obtainable. The muscles of each calf and each peroneal nerve were sensitive to pressure. No atrophy was noted and sensation was normal.

Diagnosis: Specific myelitis.

Microscopical examination: No evidence of specific changes was noted. In the lumbar segment the anterior and lateral columns were normal except for a few spaces where fibers had dropped out (*Lueckenfelder*). In the posterior columns the areas of sclerosis and rarefaction were small and seen as small islands surrounded by normal fibers. The fibers contiguous to the septum were intact. Normal fibers were found in the periphery, the root entrance zone, and bordering the gray commissure. In the areas of sclerosis were found small vessels with thickened walls; their lumina were distended with blood. In the thoracic segment the islands of sclerosis in the posterior columns were more numerous, the areas between them showing the *Lueckenfelder*. In this section there is also involvement of the lateral tracts and direct pyramidal tracts, characterized by *Lueckenfelder* and sclerosis. In the cervical segment the sclerosis in the posterior columns is more extensive than in the other segments and has the form of a triangle. Around the root entrance zone the *Lueckenfelder* was noted, a similar dropping out of fibers was seen in the lateral and anterior fields of white matter. The crossed pyramidal tracts are not so markedly involved as in the thoracic portion. The cells in the gray matter showed considerable pigmentation; the Nissl bodies were broken into fine granules and the nuclei were pushed to the side. In the medulla above the decussation the *Lueckenfelder* was noted in the lateral tracts. No changes were detected in the pons. In the brain the blood-vessels showed congestion, the cells were markedly pigmented, the nucleus in some had been pushed to the side, the cell bodies were swollen. With the Nissl stain some of the Betz cells showed loss of staining power.

Summary: Sudden onset with weakness and paresis in the lower limbs; patellar tendon reflexes were exaggerated on each side, also patellar and ankle clonus. Paresthesia in lower limbs. The duration of the illness was three months. Clinical diagnosis was specific myelitis. The lesions were symmetrical and most marked in the lower cervical and upper thoracic areas. The early character of the process was noted by the small areas of sclerosis, though the changes were far more extensive than one would expect to find in an illness of three months' duration. The lesions were found as low as the lumbar region and as high as the medulla. The changes in the brain cortex corresponded to the findings of other observers in their studies of anemic states; namely, congestion, pigmentation, chromatolysis, and changes in size and shape of the cells.

Pathological diagnosis: Combined sclerosis.

Case VI.—M. D., in the service of Dr. Pickett, female, aged forty-eight, weaver, was an alcoholic at times. Previous medical and family history was negative. Two years ago the patient stated that she was standing on the street waiting for a car and her feet became frosted. From that time on they felt numb. After about a year she began to have pains in the lower extremities and an increasing weakness. At present she complains of numbness, pain, and loss of power in the lower limbs. The pupils were slightly unequal, the left being a little larger than the right; they reacted to light, accommodation, and convergence. The extraocular movements were normal. Face wrinkled equally well on both sides. The tongue was protruded in the median line. The patient could not stand or walk. Upper extremities: The grip was not very strong but about equal on each side. The biceps tendon reflex responded slightly. No atrophy of any muscle was noted. Lower extremities: The patellar tendon reflexes were absent on both sides, even on reinforcement; also the Achilles tendon reflex. Babinski's sign was doubtful. The left leg was smaller than the right. Sensation to touch was diminished in the lower extremities. There were no areas of anesthesia and no loss or diminution of pain sense. The lower limbs were contracted, all voluntary power was lost and passive extension almost impossible. The mental state was emotional.

Microscopical examination: Lumbar segments: In the posterior columns the sclerosis was triangular in shape with its base at the periphery. The fibers around the gray commissure were intact as well as those forming the root entrance zone and oral field at the septum. The lateral portion of the white matter was involved, the remainder of the white matter was normal. In the thoracic region the posterior columns were extensively involved; the area was triangular in shape. The crossed and direct pyramidal and posterolateral tracts were extensively and symmetrically involved. In the cervical regions there was complete sclerosis of the posterior columns except for a few fibers of the root entrance zone. The sclerosis in the lateral and anterior white matter was marked. *Lueckenfelder* were found in the pyramidal tracts and in the lateral tract. In the medulla at its upper portion a similar involvement was found in the pyramidal and direct cerebellar tracts. No changes were found in the pons. In the cortex the cells showed pigmentation, chromatolysis, and swelling. The nuclei had migrated to the side. The blood-vessels were distended and congested and in one portion a small area of thrombus, hemorrhage, and softening was noted. In the cord and especially in the posterior columns numerous small vessels were seen with sclerosed walls and distended lumina. In a few areas small hemorrhages were noted. The cells in the gray matter showed pigmentation and some chromatolysis.

Summary: In Case VI there were weakness and paresthesia in the legs. Left pupil was larger than the right. There was no cranial nerve involvement; absence of all reflexes. Sensation to touch was diminished in the lower extremities. Duration of the illness was about two and a half years.

Microscopical examination: The areas of degeneration were extensive and symmetrical, extending from the lumbar region to the upper border of the medulla. The most extensive involvement was in the lower cervical and upper thoracic areas. The chief features were the marked degree of degeneration and the extent to which the brain was affected.

Pathological diagnosis: Combined sclerosis.

I wish to take this opportunity to thank Dr. Spiller for the privilege of reporting these cases and for his kindly criticisms.

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PURULENT MENINGITIS SECOND TO PANSINUSITIS; OPERATION; RECOVERY.[1]

By Wm. G. Shields, Jr., M.D., Wm. G. Spiller, M.D.,
and
Edward Martin, M.D.,
of Philadelphia.

The case described in this paper is deemed worthy of publication because it was one in which recovery occurred after operation upon the brain, when the lesion found was purulent meningitis.

REMARKS BY DR. SHIELDS.

G. P., aged fourteen years, a well-developed and well-nourished girl, who has always been healthy and led an active athletic life, as well as a studious one. Family history is good in every respect.

Five years ago developed a right-sided otitis media, which promptly responded to treatment and, with the exception of an acute attack two years ago, has not bothered her since.

Two weeks before the beginning of this illness the patient struck her head with a hanging plant on that portion of the scalp corresponding with the region of the fissure of Rolando on the right side. She did not lose consciousness, and, aside from complicating the diagnosis, the blow was unimportant, as later examination of the shaved scalp and skull showed that this trauma was not a causative factor in the present condition.

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[1] Read before the College of Physicians of Philadelphia, April 7, 1909.

I was first called to see the patient on the evening of February 21, 1909, when she had a temperature of 104° F. and a pulse of 124, complaining of general headache and aching pains over the body, with some dull pain in the right upper jaw and right ear, from which there was no discharge, and which local examination showed to be apparently normal. There was some slight pharyngeal catarrh and mucopurulent nasal discharge, but the latter had been present for some weeks preceding this time. The heart, lungs, and abdomen were negative. As a sister and brother had recovered from attacks of influenza, in the same house, just preceding this illness, the patient was treated for an influenzal infection.

On February 22 the temperature came down to 102.8° F. and pulse 120, but patient seemed to experience no relief from the general aching pain, and in addition had more severe headache, which was confined to the frontal and temporal regions of both sides. An edema of the right upper lid developed that afternoon and gradually increased until she could barely separate the lids sufficiently to show any of the eyeball. There was no rigidity of the muscles of the neck. The pupils were equal and slightly contracted, but reacted to light and in accommodation.

On February 23 the edema of the right eyelid increased and the lid was purplish in color. The headache was mostly frontal, and there was considerable tenderness at the inner canthus of the right eye, with slight supra-orbital tenderness of the right side. The temperature ranged between 103° and 104° F., and pulse 116 to 124, until 5 P.M., when temperature rose to 105.2° F., and patient became very restless. Two hours later she developed a convulsion, commencing with twitching of the lips and rapidly becoming absolutely general, accompanied by opisthotonos, rapid, stertorous respiration, cyanosis, rapid, small, irregular pulse, and biting of the tongue. It lasted for seven minutes, and patient remained in stupor for over an hour afterward. For some hours after regaining consciousness she complained of headache, and seemed confused and drowsy. As has been stated, the temperature remained high during the early part of the illness and did not respond in the least to repeated sponging. The nasal discharge having somewhat lessened, the edema of the right upper lid persisting, and tenderness over the region of the right frontal sinus having increased during the night, together with persistent hyperpyrexia, Dr. Hearst was asked to examine the accessory sinuses of the nose, and found a right frontal sinusitis, which seemed to be draining quite well. Fourteen hours after the first convulsion the patient developed another of exactly the same character, only lasting slightly longer. She was removed to the Germantown Hospital, with a view to the probable necessity of opening the frontal sinus. At this time it was thought that the convulsive seizures were probably due to the hyperpyrexia and toxemia of the infection, together with an acute nephritis which had developed and was shown by the presence of hyaline and granular casts and albumin. The Widal reaction was negative, and a leukocyte count showed 13,800. Culture of the nasal discharge showed streptococci. Heart, lung, and abdominal examinations gave negative results. Dr. Hearst and Dr. Bliss, who also saw the case, decided that in view of the free drainage through the nose and improved local appearance, the condition of the right frontal sinus was not serious enough to warrant opening it from without.

There was some intolerance of light and sound, with excessive irritability and restlessness, but no rigidity of the neck. The right upper lid was still edematous, and there seemed to be some ptosis, but as long as there was nothing abnormal with the muscles of the eyeball, it was thought that the edema could account for the ptosis. Patient complained of considerable nausea at this time. There was increasing pain in the right frontal and temporal regions, which was somewhat relieved by making continuous pressure with the hand over those regions. She complained of extreme weakness, and at times her thoughts were slightly confused. Temperature, 103.4° to 101° F. Pulse, 122 to 92.

On February 27 the left foot was seen to be extended and inverted. It could be flexed by the examiner, and there was no ankle clonus, but patient was unable to flex the foot for herself. No Kernig's sign on either side. Temperature, pulse, and general condition about the same, but she was still more nervous and irritable, with increasing intolerance of light and sound, and insisted on having pressure made continuously over the right frontal and temporal regions.

On February 28 Dr. Spiller saw the patient for the first time, and will describe the results of his subsequent examinations.

REMARKS BY DR. SPILLER.

Notes made February 28 are as follows: The movements of the lower limbs are not made with great force; the right lower limb is moved almost normally. The left lower limb is moved with a fair degree of power at the hip in flexion, but she is almost unable to make the slightest movement of extension of the left leg on the thigh, and has no movement in the left foot. The left foot is drawn inward, but can be straightened by the examiner without difficulty, although the foot will not stay in the normal position. Ankle clonus is persistent on the left side and resembles organic ankle clonus in every particular. The clonus will persist so long as the foot is held flexed on the leg. No ankle clonus on the right side is obtained, and there is no indication of it. The patellar reflexes are about normal, equal on the two sides, possibly a little diminished, certainly not in the least exaggerated When she is encouraged to make effort to extend the left leg on the thigh she seems to make a little more effort, but it is impossible to get her to appear to try, even by the use of suggestion with the faradic current. The response from the left external popliteal nerve is normal. She said the left hand was not so strong as the right, but the grasp of each hand is fully normal. The biceps and triceps reflexes are normal on each side. Sensations of touch and pain are normal in the lower and upper limbs. She has no involvement of the facial nerves. The tongue is normal, but much coated. She still has pronounced ptosis of the right upper lid, which was attributed to the previous swelling. Irritation of the sole of the left foot repeatedly produced flexion of all the toes, including the big toe, but occasionally the left big toe was extended. No Babinski was obtained on the right side. The condition of the nose and frontal sinuses has improved, the fever is less, and the child seems better than she was a few days ago. The marked weakness below the knee, as compared with the slight weakness above the knee on the left side, is striking. While the girl could flex the leg on the thigh, she could not in 'the least extend it. The ankle clonus, with normal patellar reflexes, and usually normal condition of the Babinski reflex, is also striking.

I saw the patient again the following day, at which time her condition had greatly changed. The examination gave the following results:

The lower part of left side of face is distinctly weak. She is unable to draw up the left corner of the mouth in showing the teeth, whereas she draws up the right corner in a normal manner. In attempting to draw up the corners separately, there is no movement of the left corner. She closes the eyelids firmly, but in resisting the opening of the lids by the examiner, she seems to have a little weakness of the left lids. Too much importance must not be placed on this, as it is difficult to get her to make the proper effort. The tongue is protruded straight. There seems to be no distinct stiffness of neck. She complains of great drowsiness and headache. The latter is frontal and bilateral, but inasmuch as she seems to wish her father to place his hand and press more deeply over the right side, it is possibly more right-sided. She is very irritable, evidently having much pain in the head. Last night she had considerable nausea, but no vomiting. She had no dizziness unless the head be raised. The right ptosis is still pronounced.

The left upper limb is completely paralyzed. She is unable to grasp at all with the left hand, and makes no attempt to move this limb. If the limb be placed in an awkward position, she moves it back with the right hand. She is entirely unable to recognize a finger or penknife in the left hand, even when fingers are closed upon the object. She recognizes objects promptly with the right hand. She made several mistakes in distinguishing the point from the head of a pin in the left hand, but made none in the right hand. Sense of position was not tested. The biceps tendon reflex is slightly exaggerated on the left side and normal on the right side. The triceps tendon reflex is about normal on each side.

The left lower limb is completely paralyzed except at the hip. There is some tendency to inward rotation of the feet, especially the left. That of the right is questionable. The patellar tendon reflex is about normal or a little exaggerated on the left side and lost on the right side. Reinforcement was not tested. Ankle clonus is still persistent on the left side. No indication of it on the right side. Babinski reflex is today absolutely typical on the left side. It is not obtained on the right side. Pain and tactile sensations appear to be normal in both lower limbs. There is slight indication of Kernig's sign on the left side.

The condition was evidently a focal lesion of the right motor cortex beginning in the centre for the lower limb, and my advice was to have an immediate operation with exposure of this region. Dr. Martin will describe the purulent meningitis found. The case brings up the important question whether the mere opening of the skull and exposure of the brain to the air may be beneficial in the treatment of meningitis in the way it sometimes is in a similar treatment of peritonitis. We would not, however, draw too sweeping conclusions from one case.

The swelling of the right eyelid might suggest thrombosis of the cavernous sinus, but such a lesion could not explain the meningitis beginning in the centre for the lower limb, and therefore near the superior longitudinal sinus. It seems probable that the infection was carried by a small vein from the nasal chambers or sinuses into the superior longitudinal sinus, and as comparatively few vessels empty into the anterior part of this sinus, the organisms might pass to the upper part of the brain before being arrested. The fibrous bands extending across the sinus would help to arrest the course of microörganisms, and the eddying of blood caused by the entrance of the cerebral veins into the superior longitudinal sinus in a direction opposite to the course of the blood in the sinus might aid in the deposit of the organisms at about the lower limb centre.

It is interesting to note that at first only a portion of the centre for the lower limb was affected in a purulent meningitis.

REMARKS BY DR. MARTIN.

This case suggested leptomeningitis secondary to what was almost certainly a frontal sinusitis and probably pansinusitis. Because of the swelling about the orbit the electrical trephine was first driven into the frontal sinus, which was found to be free of pus, though showing the evidence of a subacute inflammation. The posterior wall of the sinus was then drilled through, and a healthy nonadherent dura was exhibited. This wound was closed with drainage, fresh instruments were taken, and a large osteoplastic flap was turned down, exposing the Rolandic area almost to the central fissure. The dura seemed healthy. On incising it and turning it down, there was a free escape of turbid fluid and a typical picture of leptomeningitis, most intense over the leg area, gradually becoming less marked when the face area was reached. A rubber wick drainage was inserted and the flap sutured in place.

Dr. Saxon, the resident physician, describes the condition following the operation:

The postoperative treatment was, of necessity, largely influenced by the irritability of the patient. Previous to the operation very little sleep was had, and afterward it was thought not advisable to disturb her for treatment. Moreover, treatments distasteful to her would cause great mental perturbation, which would last for hours. In consequence of this, such treatment as fractional doses of calomel, etc., had to be abandoned.

During the first five days following operation the treatment was directed mainly toward an effort to get free purgation, to induce sleep, and to correct the profound toxemia which existed. The first of these indications was by no means easy. As a result of the morphine which had to be given previous to the operation, there was a marked adynamia of the intestinal tract, and no bowel movement had occurred for two days. It was impossible to give castor oil or other bulky purgative. Because of this, calomel was given in three grain doses daily for three days, and was in each instance followed by liquor magnesii citratis, f℥v. Free purgation did not result until the fifth day following operation, and to accomplish this frequent enemata were given, and recourse was finally had to oleum tigli, ♏ij, and pulveris jalapi compositus, gr. xx. From the fifth to the twelfth day daily courses of calomel were given.

For the irritability and sleeplessness, morphine sulphate, gr. $\frac{1}{12}$ p. r. n., up to gr. $\frac{1}{2}$ in the twenty-four hours, together with sodium bromide and chloral, were used in large doses.

The treatment directed to the toxemia was continuous proctoclysis.

Because of a low tolerance to salt solution, no more than two and one-half liters in the twenty-four hours could be given. Great care was taken in this part of the treatment, because it had to be carried on in such a manner as not to disturb the patient; to this end an apparatus described in the March, 1908, number of *Annals of Surgery* was used. The proctoclysis was continued for a period of ten days.

It is needless to say that much of the treatment was purely symptomatic, and need not be mentioned here; but too much emphasis cannot be laid upon the efforts directed toward the establishment of free elimination, and this was accomplished by liberal purgation, continuous proctoclysis, and the frequent administration of water by mouth.

Throughout the mental crisis the diet was liquid. As soon as this subsided, however, the patient's tastes were largely gratified, and she was given wholesome foods; pastries and sweets only were denied.

With the onset of convalescence the treatment was directed to the paralyzed muscles. The weaker groups, such as the anterior tibial and the extensors of the wrist and fingers, were given support to prevent contractures and tension, and they were exercised daily by the slowly interrupted, secondary current. Not more than ten contractions daily, of each group, were given.

When the case became ambulant, the treatment was mainly hygienic.

An examination made March 25, 1909, by the consulting physicians gave the following results:

The left foot can be moved a little, turned slightly inward, but the voluntary movement is slight unless the whole lower left limb is moved. There is much more power at the left knee and hip. Tactile and painful sensations are normal in left lower and upper limbs, and in the left side of the face. Ankle clonus and patellar clonus are pronounced on the left side. Patellar reflex much increased on the left side. The left hand cannot be moved at all except in associated movements. When she tries to move the left lower limb, or left shoulder, or to grasp with the right hand, the left hand is drawn upward by flexion at the elbow. She has slight power at the left shoulder, none at the elbow; the left biceps and triceps reflexes are exaggerated. Recognition of objects in the left hand is very imperfect, and sense of position is not entirely normal, though little affected. The left side of face in the lower part is still a little weak, but much power has returned. The left eyelids are not closed with quite the same power as are the right. The tongue is protruded straight. The general condition is excellent. Since this examination much improvement has occurred in the hemiplegia.

NOTE.—Since the above report was made complete recovery has ensued. The child has no trace of the hemiplegia, or any symptoms suggestive of her former serious condition, except that occasionally she has a left-side convulsion usually of slight intensity, and more frequently attacks of mild left-side paresthesia, usually confined to the left hand.

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DISSOCIATION OF SENSATION IN THE FACE OF THE TYPE INVERSE TO THAT IN SYRINGOMYELIA. THE RECOGNITION OF CONTACT IN THE EYEBALL THROUGH THE FIBRES OF PAIN.[1]

By WILLIAM G. SPILLER, M.D.,

Professor of Neuropathology and Associate Professor of Neurology in the University of Pennsylvania.

A CLINICAL case, even without careful study of the lesion producing the symptoms, occasionally affords a valuable contribution to knowledge. The following report is deemed worthy of publication because it throws some light on the position of the sensory fibres of the trigeminal nerve within the pons, and indicates the fibres by which contact with the eyeball is recognised.

Ziegler, aged 37 years (1909), was referred to Dr John H. Musser, November 3, 1907. He denied syphilitic infection. He had been in good health until two years previously. He then had for a time dull pain in the lumbar region, difficulty in voiding urine, and constipation. The relation of these signs to his later condition is uncertain. In April 1907 he became easily tired. In August 1907 he noticed a roaring noise in the left ear, and hearing became impaired; at the same time headache and vertigo developed, and he began to be uncertain in walking, especially in going down stairs. He had a burning sensation which began at the left corner of the mouth, extended over the left eye, and then down the entire right side of the body. He described this sensation as a scalding feeling. The headache was sharp, and was located in the occipital region.

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[1] Read before the Philadelphia Neurological Society, Oct. 22, 1909.

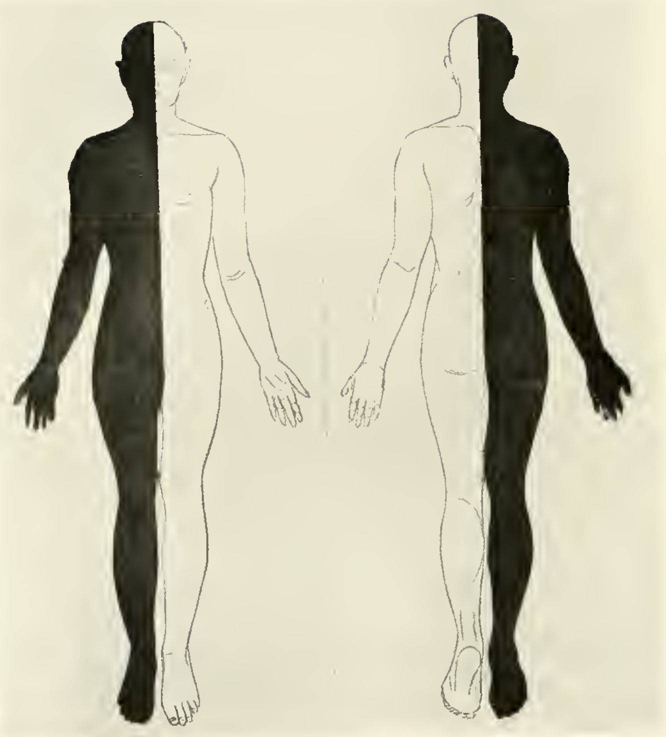
These symptoms gradually increased in severity. About two weeks before admission to the hospital the man noticed a drooping of the left corner of the mouth. The vertigo became worse. At the examination his gait was somewhat hesitating, and he had a tendency to go to the left. He had some rotary nystagmus and decided lateral nystagmus, and paresis of the left external rectus muscle. The patellar reflexes were increased. Dr G. E. de Schweinitz found no changes in the optic discs and the visual fields for white and red were normal. Dr B. A. Randall found incomplete but extreme deafness for all tones in the left ear, with reduced bone conduction, a little evidence of middle-ear disease. The hearing in the right ear seemed to be normal.

He was seen at this time by Dr C. K. Mills, and it is not surprising that a tumor of the left cerebello-pontile recess was diagnosed. The left nerve deafness, the left facial paresis, the left external rectus muscle paresis, and the ataxia were strong points in favour of this diagnosis, and no weakness was present. One sign, however, may not have received sufficient attention, it was the sensation of burning beginning in the left side of the face and extending over the entire right side of the body. This sign indicated involvement of the central sensory tracts, and with the integrity of the central motor tracts would seem to point, even at this early period of the disorder, to a lesion behind the pyramidal system.

Dr C. H. Frazier exposed the left cerebello-pontile recess, November 16, 1907, but no tumour was found. A gush of cerebro-spinal fluid seemed to relieve intracranial pressure, and following the operation improvement in many of the symptoms occurred.

The man returned to his home, and was readmitted to the hospital January 1, 1909. In May 1908 he began to vomit about once daily; this continued about one month, and then the vomiting occurred about once a week until the time of his readmittance. His speech became "thick," indistinct, and a peculiar rolling of the head from side to side began.

Dr de Schweinitz reported, January 20, 1909, as follows:—O.D. vision, 4/25; O.S., 4/20. There is complete paralysis of



FIGS. 1 and 2.

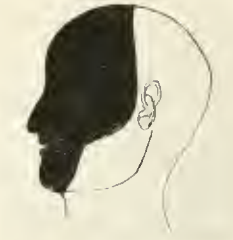


FIG. 3.

To illustrate Dr Spiller's Paper

the left external rectus muscle. O.D. is congested, and there is abrasion of the corneal epithelium. The right conjunctiva and cornea are anæsthetic, but not the left. There is no papillœdema, but a little œdema of the fibre layer of the retina, especially on the nasal side. Left eye: no papillœdema, and retinal circulation is about normal, but outer half of each disc is exceedingly pallid, indicating atrophy. Field of vision with hand unlimited.

The man was seen at this time by Dr Alfred Reginald Allen, who diagnosed a lesion of the left tegmentum of the pons.

I give condensed my notes taken February 11, 1909. They represent the condition at the present time, as no essential change has occurred. He has been frequently examined by me during the past nine months, and is now in the service of Dr Mills.

Speech is very peculiar, indistinct, thick, somewhat explosive, monotonous, bulbar; each word is distinctly separated from every other word, but speech is hardly scanning. There is distinct up and down nystagmus in looking upward or downward, and lateral nystagmus in looking to either side; but no paralysis of associated ocular movements. The tongue is protruded straight. He has entire loss of sensation for heat, cold and pain on the entire right side of the body, including the face and limbs. He has slight impairment of sensation of heat but not of cold over the left forehead. Tactile sensation is entirely normal over the entire right side in the parts in which temperature and pain sensations are lost, and sense of position is promptly recognised in the right hand. Tactile sensation is entirely lost in the left side of the face in the distribution of the fifth nerve, but pain and temperature sensations are normal in this region, excepting the slight impairment of heat sensation in the left forehead. The tactile loss does not extend over the scalp behind a line drawn vertically to the ear, or into the distribution of the cervical nerves on the left chin. Deep pressure is felt below the left eye, possibly from disturbance of the adjoining sensory area, but is not felt above the left eye. When the mouth is opened the jaw deviates markedly to the left. The left temporal and masseter muscles are completely paralysed, and there is much flattening in the portion of the face normally occupied by these muscles, presumably from atrophy The soft palate moves normally on each side. Finger to nose test shows extreme inco-ordination in each hand, equal on the two sides. Grasp of each hand is fully normal. Biceps and triceps reflexes are about normal on each side. The upper and lower extremities are not weak. Patellar reflex is exaggerated on the right side, but is normal on the left side. Persistent ankle clonus is obtained only on the right side, but there is no positive Babinski reflex. The man can stand only when supported on each side, and can take steps, but movements of the lower limbs are extremely inco-ordinated.

He is perfectly intelligent, and the tests are made with certainty because of his ready responses. The paralysis of the left seventh nerve varies somewhat in the different examinations. Taste sense was tested several times, but the results were not satisfactory; it is probably intact on both sides of the tongue. The eyes were examined in Dr de Schweinitz's clinic by Dr Williams, July 7, 1909, and he reported as follows:—Temporal sides of discs are pale, whole retinæ are blurred and œdematous, as are also the edges of discs; there is no papillœdema.

At the present time, when a test-tube containing hot or cold water is placed upon the right eyeball the man is entirely unable to recognise the changes of temperature. The contact of the head of a pin he recognises very imperfectly if at all in the right scleral conjunctiva, but somewhat in the right cornea. Sensations of heat or cold are promptly recognised in the left scleral conjunctiva or cornea, and contact with the head of a pin is promptly felt in the left eyeball. Irritation of either eyeball does not produce a reflex of the lids, because of the great impairment of sensation in the right eyeball, and of the facial paresis involving the orbicularis palpebrarum on the left side.

Dr C. P. Grayson gives the following report of the laryngeal examination:—October 21, 1909. The throat is not easily examined, not only because of the abundant salivary secretion, but also because of the patient's inability to expectorate it. Although I was unable to see the vocal cords, I saw perfectly well the arytenoids and a portion of the ventricular bands. On phonation the excursion of the right arytenoid is not only complete but exaggerated, since it moves beyond the median line in its attempt to meet the left, the movement of which is so restricted that it shows scarcely more than a slight twitch. Without being positive, I am confident that the left cord is paralysed.

Two groups of symptoms in this case are of special interest: (1) The dissociation of sensation of the syringomyelic type (preservation of tactile sensation and of sense of position, loss of pain and temperature sensations) on the entire right side of the body, including face and limbs, with an inverse type of dissociation of sensation to that of syringomyelia (preservation of pain and temperature sensations, except of heat sensation over the left forehead, with loss of tactile and pressure sensations) strictly confined to the distribution of the left fifth nerve, and with loss of function of the motor portion of the left fifth nerve.

(2) The greatly impaired or lost ability to recognise contact, heat and cold sensations in the right eyeball, on the side in which tactile sensation is preserved in the face as well as elsewhere; with the preservation of recognition of contact in the left eyeball on the side in which touch sensation is entirely lost but pain sensation is preserved, in the distribution of the left trigeminal nerve.

Inasmuch as the symptoms in this case have not changed in any important details at least during the last nine months, and papillœdema has not developed, the diagnosis of tumour is questionable. The symptoms in association with marked pallor of the temporal sides of the discs suggest multiple sclerosis. Softening or cavity formation of the pons is at least possible, but not probable.

The lesion seems to be one that attained its full development at least nine months ago, and is now very little if at all progressive. Its situation must be in the left tegmentum of the pons. It implicates the central fibres of pain and temperature sensations coming from the entire right side of the body, probably those from the trunk and limbs being contained within the tractus tecto- et thalamo-spinalis or Gowers' tract within the left side of the pons, and those from the face possibly in the central tract for the face fibres nearer the raphé, as the central tract of the trigeminal nerve in the medulla oblongata is believed by some to be situated a little in front of the hypoglossus nucleus.

The lesion involves the left sixth nerve and the left acoustic nerve or its central tracts passing to the right side of the brain stem. It involves completely the motor portion of the left trigeminal nerve in or near the motor nucleus; it involves also all the fibres of touch in the left trigeminal nerve, and slightly those of heat coming from the left forehead. The sensory root of the trigeminal nerve divides soon after entering the pons; some of its fibres pass a short distance upward to terminate in the sensory nucleus within the pons, whereas a large portion of the fibres from this root passes downward into the medulla oblongata near the periphery of the pons. The ascending fibres probably are those of touch, and are implicated, together with the motor fibres of this nerve, in the lesion; whereas the fibres which descend near the periphery of the pons probably are concerned with temperature and pain sensations, and these evidently are not implicated in the lesion. The central fibres from the nucleus accompanying the spinal root of the trigeminal nerve cross the raphé at different levels, and are not seriously implicated in the lesion. Whatever explanation may be offered for the peculiar sensory disturbance in the distribution of the left fifth nerve, it seems clearly demonstrated by this case that the tactile fibres separate soon after entering the pons from the pain and temperature fibres of this nerve.

Babinski[1] has stated that he has been unable to find any carefully studied case of crossed bulbar anæsthesia of the inverse type to that of syringomyelia, *i.e.* preservation of temperature and pain sensations and loss of tactile and muscular sensations. It is strange, as he remarks, that in bulbar anæsthesia this inverse type is never seen. The theory of different tracts for the different forms of sensation is founded on the examinations of spinal cord lesions, and he concludes that it is incorrect so far as the brain stem is concerned. This case studied by me seems to indicate that the fibres of touch and muscular sensations (pressure) in the trigeminal nerve separate immediately on entering the pons from the fibres of temperature and pain sensations in a manner similar to that of posterior root fibres. It would indeed be strange if after decussating through the raphé of the pons and medulla oblongata they should reunite to form a single sensory tract.

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[1] Babinski, *Revue Neurologique*, 1906, p. 1177.

It is possible that some other case may be on record in which a lesion of the pons has caused a type of dissociation of sensation the opposite to that of syringomyelia, as has occurred in this case I have studied, but I do not know of it.

The peculiar behaviour of the eyeballs in regard to recognition of contact needs explanation. On the right side on which tactile sensation of the face was normal but pain sensation was lost, contact of the eyeball with the head of a pin was so imperfectly recognised as to make it doubtful whether it was recognised at all. On the left side in which tactile sensation of the face was lost but pain sensation was preserved, contact with the eyeball was promptly recognised. The case would appear to justify the statement that recognition of contact in the eyeball depends more upon the fibres of pain than upon those of touch. When a foreign body rests upon the eyeball the sensation is chiefly one of discomfort or pain.

Sherrington makes the following statement in regard to this matter :—" The cornea and ocular conjunctiva are rich in pain spots, but have no touch spots ; so also the glans penis. The eyelid sweeps over the cornea without being felt." [1]

" There is a difference of opinion as to whether any touch can be felt on the cornea without pain. Nagel has pointed out that touches can be felt on the conjunctiva and cornea without any painful or disagreeable quality in them. It is the ease with which the corneal endings provoke pain which is denoted by the term ' pain spots ' as used in the above paragraph."

The absence of touch spots in any portion of the body is interesting. Rivers and Head [2] do not refer to the cornea, but they state that the glans penis is not sensitive to cutaneous tactile stimuli, though pressure is correctly appreciated and localised with fair accuracy. It appears to me as probable that a similar condition exists in the eye. The patient studied by me seemed to recognise occasionally contact in the right eye if some pressure over the eyeball were made with the head of the pin.

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[1] C. F. Sherrington, " Text-Book of Physiology," by E. A. Schäfer, vol. ii., p. 987, 1900.

[2] Rivers and Head, " Brain," vol. xxxi., 1908.

Description of Figures.

Figs. 1 and 2.—The shaded areas indicate the loss of pain and temperature sensations, with preservation of tactile sensation and sense of position in the hand.

Fig. 3.—The shaded area indicates the loss of tactile and pressure sensations, strictly confined to the distribution of the fifth nerve, with preservation of pain and temperature sensations, except impairment of heat sensation over the left forehead.

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SYPHILITIC PARALYSIS OF THE TRIGEMINAL NERVE.

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AND

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The trigeminal nerve is seldom paralyzed without implication of other cranial nerves, and the suspicion is occasionally entertained that syphilis may be the cause of this paralysis. It is important to know the frequency of syphilitic disease of this nerve, especially of paralysis confined to this nerve. We have several times found bilateral degeneration of the trigeminal nerve, including the spinal root, in tabes.

Oppenheim[1] refers to a long list of authors who speak of implication of the trigeminal nerve in cerebral syphilis, but apparently he is referring chiefly to the clinical manifestations, although he mentions that he has had confirmatory pathological findings. In one of his cases the spinal root of this nerve was affected, as it was also in a case reported by Brasch, and in one reported by F. Pick. According to Uhthoff's statistics, the trigeminal nerve seems to be implicated in syphilis as often as the abducent nerve, that is, in 14 per cent. of all cases of cerebral syphilis. Usually the implication of this nerve is unilateral, although exceptions to this rule occur (Leudet and Labarrière, Hutchinson). The sensory branch is much more frequently affected than the motor. Pronounced paralysis of the muscles of mastication with atrophy has occurred very exceptionally (v. Ziemssen, Loewenfeld, Oppenheim).

Nonne[2] states that Huguenin found gumma of the Gasserian ganglion; Balfour, gumma of the sella turcica, with pressure on the trigeminal nerve; and Uhthoff, in 37 necropsies in which the trigeminal nerve was found diseased, observed involvement of this nerve alone only in four. Degeneration of the spinal root of the trigeminal nerve in cerebral syphilis he states has been observed by Oppenheim, F. Pick, Brasch, and Cassirer. In all these cases the involvement of the nerve was unilateral. In Pick's case the trigeminal nerve was implicated in a gumma, in Cassirer's the cause was intense basal meningitis, in Oppenheim's and in Pick's the Gasserian ganglion was diseased.

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[1] Oppenheim. Nothnagel's System of Medicine.

[2] Syphilis und Nervensystem, second edition, p. 233.

Recently one of us (Spiller) has seen a case of cerebral syphilis in consultation with Dr. S. R. Crothers, in which left hemiparesis was associated with partial palsy of the third nerves and weakness of the motor branch of the right trigeminal nerve. The sensory branch of the trigeminal nerve was not affected. When hemiplegia from syphilis is associated with paralysis of cranial nerves, two lesions usually are found as the causes of the symptoms: a basal syphilitic meningitis producing the cranial nerve palsies, and arteritis producing occlusion of a bloodvessel with softening in the region of the internal capsule.

Five years ago a case of syphilitic disease of the fifth nerve was studied clinically by one of us (Spiller) and later the microscopic examination was conducted by us conjointly. The extraordinary fibrillary tremors of the right masseter muscle indicated that this muscle was in a condition of acute degeneration. So far as could be determined from the history and the examination, the only cranial nerve affected was the fifth on the right side, although the eyegrounds were not examined; and this fifth nerve palsy was associated with recent right hemiplegia implicating the right side of the face as well as the limbs. The palsy of the fifth nerve was inferred from the intense fibrillary contractions confined to the masseter muscle, as the man was too unconscious to respond to tests, and confirmation was obtained by the microscopic examination. No statement is made as to fibrillary contractions in the right temporal muscle, although they may have been present. The palsy affected the motor branch, and this is more uncommon than is implication of the sensory branch, but from the history it is probable that the sensory branch did not escape, as it is stated that he had pain on one side of the face. No gross lesion explanatory of the fifth nerve palsy was recorded in the notes taken by the pathologist (Dr. Alburger) who performed the necropsy, and it is probable, therefore, that it was caused by the intense syphilitic meningitis found by the microscope.

W. L., laboratory number 111, male, aged thirty-five years, colored, was admitted to the service of Dr. Stengel, March 2, 1904. About one year previously he had a stroke involving the left upper limb, which was partially paralyzed, and from this he never entirely recovered. He always dragged the left lower limb slightly after this attack.

He had suffered from "neuralgic" headaches, as he called them, a number of years. The pain was usually on one side of the face and head. It is presumable that it was on the right side, although it is not so stated. He seemed well four days before admission to the hospital. On the evening of February 29 he complained of headache, but seemed well the following day and went to work, but had to stop work because of the headache; about six o'clock his friends noticed that he could not speak, although he was still able to walk about, and he ate moderately at the evening meal. He retired soon after dinner; on the following morning he was unable to get out of bed, and was unconscious; he could not answer questions, and could take neither water nor food.

Notes taken by Dr. Spiller shortly after his admission to the hospital are as follows: The patient is unconscious. When he is called by name he opens his eyes, but makes no sign of understanding what is said to him. He does not open his eyes very widely. The eyeballs are not deviated. He is able to look upward and to the left, but it is impossible to test the movements of the eyeballs carefully. He closes the eyelids firmly on each side, and seems to have a little flattening of the lower part of the right side of the face.

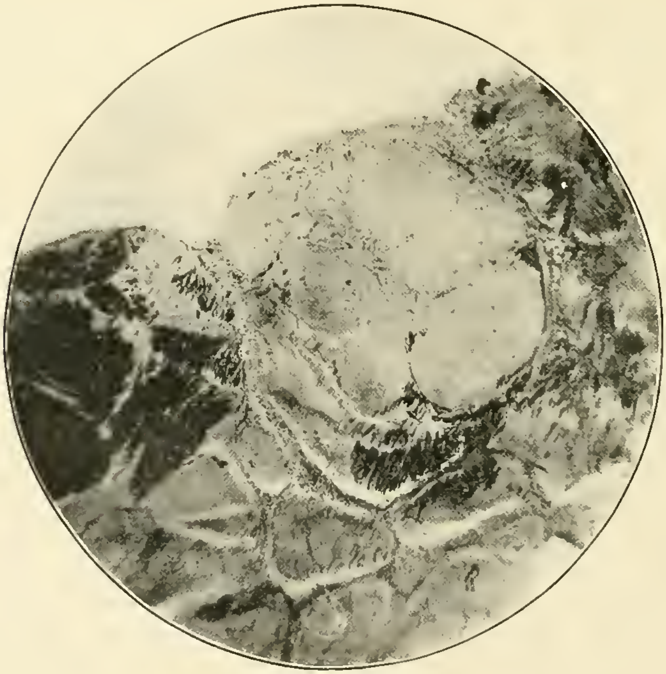
When he is told to show his teeth he attempts to do so, and the lower part of the face is moved only on the left side. He has, therefore, paralysis of the lower part of the right side of the face, but it is impossible to get him to show the tongue. The right side of the face does not contract when it is stuck with a pin, but marked contraction occurs on the left side of the face. Very intense fibrillary contractions occur continually in the right masseter muscle. The contraction of the masseter muscles cannot be tested, as it is impossible to get the man to make chewing movements.

Upper limbs: He raises the left upper limb freely above the head. The right upper limb is in slightly increased tonicity. The biceps-tendon and triceps-tendon jerks and wrist reflexes are exaggerated on the right side. The corresponding reflexes on the left side are about normal. When the right upper limb is stuck with a pin very little attempt is made to move it, but he tries unsuccessfully to find the source of irritation with the left hand. Sensation for pain is probably preserved in each upper limb. The left upper limb is somewhat spastic.

Lower Limbs: The right lower limb is paralyzed. He can flex the right thigh slightly when the limb is stuck with a pin, and can flex the right knee slightly. The left lower limb is moved freely. The pinprick is perceived well in each lower limb. The right patellar reflex is exaggerated; the left is about normal. The Achilles jerk seems to be about normal on each side. A slight indication of ankle clonus is obtained on the right side. Babinski's reflex is not obtained on the right side, the toes not being moved at all. It is obtained on the left side, all the toes except the big toe being distinctly moved upward. The right lower limb is somewhat spastic, the left is about normal.

The man died shortly after admission to the hospital.

An area of softening was found in the left temporal lobe. Sections stained with the hemalum and acid fuchsin showed an intense round-cell infiltration and numerous fatty granular cells in this region. This lesion was probably the cause of the right hemiplegia. Slight degeneration was found in the left anterior pyramid and marked degeneration in the right anterior pyramid by the Marchi stain. Probably sufficient time before death had not elapsed for intense degeneration of the left anterior pyramid, detectable by the osmic acid, to occur.



Section of the roots of the right trigeminal nerve, showing an area almost completely degenerated (Weigert stain.)

Sections of the roots of the right fifth nerve showed much degeneration when stained by the Weigert method or by acid fuchsin with hemalum, and in some parts of the sections the nerve fibers had entirely disappeared. Recent degeneration was revealed by the Marchi method in the right fifth nerve with in the pons and within the spinal root of this nerve. The pia over the pons, optic chiasm, and optic nerves presented intense round-cell infiltration. The left crossed pyramidal tract was more degenerated than the right.

We are indebted to Dr. Allan J. Smith for the photograph.

Reprinted from the New York Medical Journal for September 3, 1910.

ALCOHOLIC AMNESIA, WITH THE CLINICAL REPORT OF A CASE.*

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Among the various forms of mental disturbance is diminution or loss of memory. This may result from different causes, an important group being due to intoxications, and the particular intoxicant concerned in the case here reported is alcohol.

Alcoholic amnesia may be either functional or organic, partial or complete, and again it may be temporary or permanent, these being terms which are self explanatory. Another important class is worthy of a word of explanation. We speak of retrograde amnesia, where the failure of memory is for a period of time immediately preceding a certain forgotten event, antegrade, where the attack is subsequent to such a time, and finally, with defective memory at both of these times, we have a condition spoken of as anteroretrograde amnesia.

Whiskey is the substance most usually consumed, though the excessive use of other drinks may result in this state. The amount of the intoxicant varies, but many of the patients have been known as hard drinkers and frequently they have begun drinking early in the morning.

Mental symptoms other than amensia are not of such common occurrence as in delirium tremens,

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*Read before the Philadelphia Psychiatric Society, May 13, 1910. The case is reported by permission from the Philadelphia Hospital for the Insane.

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and in the grave alcoholic insanities such as the delusional, paranoid, and pseudoparetic forms.

The failure of memory may appear abruptly, but it usually comes on gradually, and recent occurrences are most often forgotten. The individual may be able to talk freely of events which have come before him a long time previous to the amnesic period. While word blindness and word deafness are not present, those impressions when received are but transitory. Hallucinations and delusions are either not conspicuous or are totally absent. An alcoholic history is usually obtainable from the patient or someone else, and the appearance of the individual may be that of a drinking man, but in the absence of these data there is one condition which should be thought of and that is the psychic equivalent of epilepsy; subsequent observation may be necessary to clear up the diagnosis.

A difficulty which at times confronts the examining physician is that of simulated amnesia. There may be embarrassing or even criminal acts to conceal and a convenient lapse of memory is at times resorted to.

With good attention the condition is generally a hopeful one, but recovery does not usually occur under several weeks, and even years have elapsed before full memory has been regained. For care and treatment these cases often require detention in an institution. They need abundant nourishment for they are underfed, indeed most of them have been for a long time substituting alcohol for food. A reconstructive tonic is always indicated, and the bowels, kidneys, and skin should be kept active for the elimination of any remaining alcohol and effete body products. Drugs and good hygiene are used to place the patient in the best possible physical condition. There is no specific for alcoholism. It is absurd to believe that will power can be supplied in bottles. Of course a patient may be kept under the influence of some drug while alcohol is being withdrawn, but there is no special drug virtue in these so called "cures," otherwise there would not be so many new ones brought forward from time to time. Psychotherapy comes in usefully. These patients often strive diligently to overcome their mental failure and therefore they should be reeducated. Later suggestion can be employed advantageously, and possibly some of the special hypnotic methods may have value. Forel, of the University of Zurich, whose experience has been as large as that of any one, reports good results from the use of suggestion. Dubois, of the University of Berne, uses psychic treatment almost to the exclusion of drugs in his management of various psychoses. We know the temperance societies hold some cases in this way; they have backsliders, of course, but unfortunately we do also.

In these times when patients frequently seek advice given by others than a physician, it is well to remember that the importance of certain medical matters is occasionally emphasized to the lay mind by persons outside of the profession, and the condition resulting from our partial neglect of a therapeutic measure may be worthy of a moment's notice. Christian science has in part become established as the result of a need; if this were not true, this cult could not have grown so rapidly. Its spread is not due solely to its having become a fad. Persons often desire and are benefited by psychic support and guidance, and if it is not given by a physician, they will turn to some one who is more obliging. No doubt, as has been charged, the leader of this so called science obtained most of her ideas of these matters from medical men (one of her husbands was a physician) but a glance at her writings shows at once she is not sharing honors; however this may be, the fact remains that important medical work has been deflected to a set of persons who are not properly qualified to assume this responsibility.

Because of the intelligence and earnest endeavor to give accurate information concerning his condition, it was possible to make a somewhat detailed study of the following case:

Patient was single, aged fifty-three, and a school teacher by occupation. Family history was negative except that his father drank quite heavily. Owing to some slight disability of his lower extremities which dated from childhood, he was given sufficient education to become a teacher. He had used alcohol moderately for some time, but for the last two years had been drinking excessively, though he still attended to his duties. Early in April of 1907 he was guilty of some trifling impropriety which gave rise to some unpleasant comment, and just about this time there was beginning failure of memory. A few days later he left his home saying he would return the following day. A note found just after his departure stated that he was unfit for teaching and that he was leaving to take a Keely "cure." Some days following, without knowing how or when, he arrived in this city and was taken into custody by an officer to whom he had just said if he were not arrested he would kill somebody or commit suicide. The time from the 12th to the 20th was spent in the detention ward. For some days he lay in a profound stupor, taking no notice whatever of his surroundings. This state was followed by marked mental confusion, a feature of which was auditory and visual hallucinations, and upon the 30th he was transferred to the Hospital for the Insane. Even at this time his memory was wanting except for a few isolated impressions, as shown by his inability to recall his own name, his occupation, or if he were married. However, he did remember his age and birthplace. From then on recollection gradually returned. On May 3d, while not sure of his name, he knew his father was dead and was quite certain he was not married. On the 4th he seemed depressed because he could not remember more, though he retained most of what he had previously recalled. By the 5th he surprised his interrogator when he announced that he could give his correct name; this he did.

Events prior to January of that year were now quite readily recalled. Careful examination revealed a poverty of memory images, especially visual, an instance of this was his inability to recall how his mother's hair looked, though he remembered that it was gray. On the 7th he said, "I can remember my boyhood pretty well now," and he was much more able to recall the visual appearance of things. By the 13th, he thought he recalled teaching school after Christmas and upon the 15th remembered many events that had taken place just before his admission to the detention ward; then followed some days of which he was totally oblivious. Many occurrences during his stay in the Insane Hospital now became firmly fixed upon his mind, and in about two months from his admission his memory had fully recovered. After complete restoration, when asked which was the more confusing, the hallucinations or the loss of memory, he replied, "The hallucinations, I think; I saw I could not trust my senses and did not know what to believe."

This was a very grave case of complete alcoholic amnesia, and I feel reasonably sure that the psychic treatment which the man received almost daily for several weeks, shortened very materially his illness. Perhaps it saved him from permanent mental disability.

1728 CHESTNUT STREET.

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COCAINE INTOXICATION.*

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There is considerable matter of historic interest associated with the drug which we know as coca. Because of its property of imparting endurance, the Incas of Peru regarded it as the "divine plant," and the natives of some of the South American countries attributed to it marvelous powers, employing it in their religious rites and sacrifices, and regarding it with both reverence and superstition. It was used, in time of war, to increase their strength and for the prevention of hunger and thirst; they also indulged in it for the pleasurable intoxication which it produced. It has been said that the natives pluck the leaves from the bushes by the wayside to chew; the leaves are selected as they are sweet and rich in the more volatile alkaloids and contain a low percentage of cocaine; those containing a larger quantity of the alkaloid have a bitter taste. After the gathering, the leaves are dried and cured with as much care as is bestowed upon the preparation of tea or tobacco. Coca is used extensively by those engaged in heavy labor, and the quantity apportioned to each worker depends upon the task he has to perform; one or two handfuls, which is equivalent to one or two ounces, is the average amount for a day's consumption. The leaves are chewed

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*Read before the Northwestern Medical Society of Philadelphia, October 3, 1910.

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with a small quantity of a limelike substance which is obtained from the ashes after the burning of certain plants, shells, or limestone. Under the sustaining influence of this drug, the natives carry heavy burdens for long distances and do other laborious work without hunger or fatigue. Strangely enough, it is said their health does not become undermined from this use, and they often live to an advanced age.

The statements concerning the sustaining power of coca would be almost incredible were they not so well authenticated by physicians and others. W. B. Stevenson, who wrote of his twenty years' residence in South America, found "that the natives in the mines, or when traveling, derived such sustenance from chewing the leaves that they frequently took no food for four or five days, though constantly working; and that they told him that with a good supply of coca they felt neither hunger, thirst, nor fatigue, and that, without inconvenience or injury, they could remain eight or ten days without sleep." Ordinarily, the use of coca gives rise to no special sensation; there may be an absence of the customary desire for food and sleep, but if an unusual demand is made upon the mind or body its influence is then felt.

We shall now consider the effect that cocaine, the active alkaloid of coca, has upon the white race. The drug is mostly used in the form of cocaine hydrochloride. Degrees of individual susceptibility vary widely; in some less than one grain will give rise to toxic symptoms, while in others, two or more grains may be taken without such disturbance; from about eight grains upward is regarded as a fatal dose, though considerably larger doses have been recovered from. A moderate dose is followed by a slight rise in blood pressure, a pleasant sensation of bodily warmth, dilatation of the pupils, exaltation, self assurance, unusual mental activity, a general feeling of well being, and increased capacity for physical effort; the individual appears somewhat excited. This is followed by a stage of depression, a loss of muscular activity, and a somewhat accelerated heart action. If the dose is a poisonous one, the respiratory movements are somewhat increased, the pupils are dilated at first but contracted toward the end, there is nausea and vomiting, an intermittent pulse, profuse perspiration, cyanosis, muscle cramps and general muscular weakness, epileptiform convulsions, and finally Cheyne-Stokes breathing. Instead of unconsciousness, a violent mania may develop. If the patient recovers, he is often in a highly nervous state for days or even for weeks after. When death ensues, it results from paralysis of the respiratory centre together with exhaustion following the severe convulsions.

The continued use of cocaine is followed by the most direful results. Before considering these, it may be of interest to attempt to explain the occurrence of these disastrous effects in the white race, since the Indians of South America seem to escape the more serious consequences. For centuries millions of these natives have used coca and had it produced in them marked mental and physical deterioration, the fact could hardly have escaped notice. In the first place we are not Indians; the greatest number of our people who become addicted to the use of cocaine are the debilitated, neurotic, and persons who are naturally perverse, and of course we have a larger percentage of these than can possibly be found among any primitive people. The use of the alkaloid being followed by such pleasant mental and bodily effects, makes it particularly attractive to us because of the many annoyances and affections attendant upon civilization, such as delayed hopes, overwork, lack of exercise, and bad air with their consequent disorders; all these find prompt relief after the use of this drug; the taking of the powerful alkaloid doubtless has a much more deleterious effect than the chewing of the leaf, many of the natives accustomed to its use live in mountainous districts, lead an active life, such as mining and the making of long journeys, and live in an invigorating climate; finally, the Indian selects a leaf that is sweet and therefore contains a small percentage of cocaine; he uses but three portions each day and usually does not increase this allowance during his lifetime.

Cocaine came into general use shortly after 1884, when its value as a local anæsthetic in ophthalmic surgery was discovered. Because of its producing exhilaration and mental exaltation, as well as being in general strikingly opposite in its body action to morphine, it was soon adopted as a means of overcoming the morphine habit; it has been used extensively for this purpose and at first appeared to act most happily, but soon it was found that the patient almost invariably became addicted to this drug, and morphine is the lesser of the two evils. Its use is unsafe in the withdrawal of morphine or in the breaking of any other drug habit. From periodical use at first, cocaine takers soon drift into its continued use, and since its effects soon wear off, it is resorted to at frequent intervals; the debilitated and neurotic take to it readily, and the poorer classes fall easy victims to its charms; the habit is common among the negroes in some parts of the south, and some criminals are users of the drug. Most cocainists use it in conjunction with morphine, often with alcohol, and rarely with chloral hydrate. It comes as a great temporary solace to those who are already exhausted from the excesses of other drugs. Persons who have found themselves drifting into its continued use, and who still have had sufficient strength of mind to throw off the habit, say that the intense longing never dies out, and being aware of its enslaving power, they are afraid to come in contact with it; I know physicians who will not keep it in their office on this account. Cocaine is the first of drugs to enthrall its devotees, luring them to an almost hopeless captivity; soon it induces physical, mental, and moral deterioration, often leading rapidly to death, but rarely to a permanent insanity. My own experience with these patients has come from a six years' residence as medical superintendent in a private hospital where addicts of some sort were always present, in the Philadelphia Hospital for the Insane, and also in the detention wards of the Philadelphia General Hospital; it may be of interest to add that but few cocaine users are transferred from the detention wards to the Insane Hospital: they seldom become chronic insane patients, but usually die in a marasmic condition or are carried off by some intercurrent affection.

The symptoms presented by cocaine addicts are those of rapid emaciation, the individual is distressed of countenance, is restless, talkative, and secretive, the skin has a pale, yellowish, withered appearance, the eyes are deeply sunken and the pupils dilated, there is muscular weakness and tremors, the extremities are cold, and there are attacks of weakness from cardiac irregularity; business, which is at first plunged into only to be left unfinished, is finally abandoned. Sometimes from the poisonous effects of the drug and perhaps also from the marasmic condition, there develops an hallucinatory phase beginning with great restlessness and distrust, from which the individual passes rapidly into a state of irresponsibility; the special senses may all be involved, vile language is often heard, and they hear persecutory threats shouted at them; they imagine to observe small black spots wandering over a light surface, see vermin of various sorts about the room or upon the clothing; a very common sensation experienced is that of insects crawling under the skin; this is known as Magnan's sign and has been termed the "cocaine bug"; this sensation of bodies under the skin is characteristic of cocainism; needlelike pricks are felt and many other painful sensations are experienced; the patient may believe these are due to the machinations of his fancied persecutors, a homicidal insanity may develop at once and he may arm himself for the destruction of his supposed enemies; accusations of marital infidelity are quite common; he has now become an exceedingly dangerous person; rarely, in this condition of mental and physical torture, he commits suicide.

When a combination of drugs is used, the diagnosis may be quite difficult. The cocainist is erratic, secretive, distrustful, and inclined to be solitary. This is usually not true of the morphine user. It differs from the alcoholic psychosis in the peculiar form of skin sensations, the more severe character of the maniacal manifestations, and in the absence of albuminuria, the administration of a single dose of cocaine will aggravate the condition, while little or no effect will result if the patient is an alcoholic. From paranoia, it may be observed that the delusions are usually less systematized, less constant, and more numerous.

If the patient is treated early, the prognosis is perhaps more favorable than in either morphine or whiskey addicts; however, except in the early stage, a permanent cure is extraordinarily rare. Those individuals who use a combination of drugs are the least hopeful.

In cases of acute poisoning where the drug has been taken internally, the stomach should be washed out at once; respiratory failure is to be met with alcohol, camphor, aromatic spirit of ammonia, and perhaps oxygen and artificial respiration; a rapid injection of normal saline solution has proved beneficial.

The chronic patients may be treated by stopping the drug at once, by rapidly reducing it, or it may be slowly withdrawn. In many cases its immediate withdrawal will be the most satisfactory, for the reason that during the gradual reduction, it sometimes happens that the patient will not remain under treatment when the discomfort incident to the partial deprivation manifests itself strongly; in those states where institutional detention is lawful, these patients are more easily managed by confinement. When maniacal symptoms are present, they usually subside when the individual can no longer obtain the poison. The suffering attendant upon deprivation is not so great as in the morphinist, but there is danger of the symptoms of collapse for some time after the drug has been withdrawn. Strychnine is a sustaining remedy; the mental distress may be relieved by hyocyamus, valerian, or large doses of bromide; the appearance of collapse should be met by the administration of cardiac and respiratory stimulants; the insomnia is best overcome by prolonged baths; good food should be given at frequent intervals.

1728 Chestnut Street.

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TUMORS AND CYSTS OF THE SPINAL CORD WITH A RECORD OF TWO CASES[1]

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The subject of tumors and cysts of the spinal cord, like that of similar lesions of the brain, has passed through several periods during the last thirty years. Neurologists and surgeons, stimulated on the one hand by the development of both cerebral and spinal localization, and on the other by improved methods of surgical procedure, took up the question of the localization of such lesions and their removal, with enthusiasm. After a few years a lull of interest occurred, the result of various causes; but during the last ten years this interest has been reawakened and increased, chiefly because of our advance in knowledge regarding the functions of the brain and spinal cord, and the greater success which has attended operations.

In the early years of the period to which reference is made, several brilliant successes, well known to all familiar with the literature of this subject, were achieved in the case of spinal tumors, both in this country and abroad. Later a considerable number of operations were performed year after year, with varying success.

One cause of renewed interest in the subject of spinal surgery based on localization, is the new outlooks which have been secured through some particular lines of recent work. The observations of Sherrington have shown that the sensory root distribution is such that the destruction of a number of roots produces anesthesia only in the domain of a single root. This is an observation which is evidently important, as has been verified in my own recent experience. A patient, for instance, with distinct indications in neural pain and cutaneous hyperesthesia of the implication of one or two posterior roots, still is found to retain sensation in the areas of the skin in which the peripheral continuances of these roots are known to be distributed.

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[1] Read at the thirty-sixth annual meeting of the American Neurological Association, held in Washington, D. C., May 2, 3, and 4, 1910.

It may require a section or destruction by disease of three, or in some cases as many as five roots in order that a definite area of anesthesia shall result.

Through the investigations of Head, and those who have worked with him or have followed him, much more accurate information is now available, regarding the distribution and course of afferent nerves both in the periphery and within the spinal canal, this knowledge having some diagnostic value.

One distinct advance is that which has come from the extension of our knowledge of diffuse and circumscribed serous meningitis as the result of contributions by such neurologists and surgeons as Spiller, Pearce Bailey, Oppenheim, Schlesinger, and Horsley. Roentgen ray explorations have occasionally been found useful, especially in determining whether or not the bone is involved in a lesion.

The first case which I shall report is one with a tumor of the lower cervical region.

Case 1.—*Intradural Tumor of Lower Cervical Region. Sharp pain in neck and shoulder for two years; weakness in left upper and later in lower extremity of the same side; moderate atrophy of the thenar and hypothenar muscles and small muscles of the hand; doubtful hypesthesia on the ulnar side of hand and forearm; sense of position affected with slight ataxia of hand; all reflexes in left upper extremity impaired; the reflexes in left lower extremity increased, with Babinski response; reflexes on the right side present and probably somewhat increased; the skiagraphs showed a shadow apparently indicating increased density of the left halves of the fourth, fifth and sixth cervical vertebræ, and also an apparent shadow outside and to the left of the fourth and fifth vertebræ. Operation revealed intradural tumor reaching from about the fourth cervical to the seventh cervical vertebra; tumor removed with uneventful surgical recovery; pain, impairment of power, and other symptoms with* *the exception of the Babinski reflex, relieved; new syndrome consisting of contracted left pupil with partial ptosis and retraction of the ball developed after operation; tumor an endothelioma.*

The patient is the wife of a physician, and was first seen by me in consultation with her husband on January 6, 1910. From this time until that of the operation, February 25, 1910, she was repeatedly examined by me. She was also examined by my colleague, Dr. William G. Spiller. The notes of various examinations made are summarized. This can be the more readily done as practically no change occurred in her symptoms during the four or five weeks she was under observation before the operation.

Mrs. S., 33 years old, had been married eight years, had two children, with a history of no miscarriages. Her father died when fifty years old as the result of disease, the diagnosis of which could not be determined. Before his final illness, he had had however attacks of dizziness with falling and unconsciousness. Her mother died at the age of sixty-seven, about three hours after an apoplectic attack.

The patient was a fine looking woman who usually enjoyed good health. She had had however several seizures which were called fainting spells, the last occurring about eight years previous to observation. In this last attack she lost consciousness, but had no spasm or other symptoms which were noted. An older sister had had similar fainting spells. She had three healthy brothers.

The chief thing to which attention was first directed two years before coming for examination, was the occurrence of pains in the back of the neck and left shoulder, which were at first and for some time thought to be rheumatic, but the pain did not respond to anti-rheumatic remedies which had been tried freely. Recently she had had spells of pain which would last five to twenty minutes and then disappear after exercise, to return again at intervals which increased in frequency. The pain in the back of the neck seemed to be somewhat different from that in the shoulder, being sharp and throbbing. During a week or two previous she had had some pain in the left upper limb below the shoulder. She had never had pain in any other part of the body.

About five or six months previous to the examination she first noticed some feeling of numbness or discomfort in the left arm, leg, and foot. She was not sure where these sensations occurred first, but believed it was in the hand. Gradually she became somewhat awkward and feeble in the use of both the left upper and lower extremities. The toes of the left foot would sometimes catch in the rugs or carpet when walking. In various ways the weakness and awkwardness in her left hand had become evident, as for instance when buttoning her own clothes or those of her children.

She had had at times difficulty in holding her urine, and she was constipated.

Examination showed her station with eyes closed to be good, but she could not stand as well on the left foot as on the right, owing apparently to weakness on the left side.

The patient wrinkled the forehead and closed the eyelids well on each side. The left corner of the mouth was not drawn up quite as easily as was the right corner. The tongue and soft palate were normal. The movements of the masseters and pterygoids were performed normally. The only evidence of motor impairment in the face was the slight difference in the power to draw up the left corner of the mouth, as compared with the right. This slight difference was probably natural in the light of subsequent findings.

The grip of the left hand was poor as compared with the right, the difference being greater than could be accounted for by her right handedness. She also had certain special conditions present in the left hand which proved to be of diagnostic importance. She could not flex the fingers of this hand forcibly so as to indent the palm as she could on the other side, and some atrophy was present in the small muscles of this hand. The little finger of the left hand tended to assume a peculiar position of abduction. It separated itself markedly from the ring finger, and the latter slightly from the middle finger, and the little finger could not be brought easily into apposition with the ring finger as upon the right side.

In a general way the movements of the left upper and lower limbs were reduced, although the only isolated loss of power was in the hand and finger movements as just described.

The finger to nose test showed slight ataxia which sometimes could be scarcely demonstrated.

Careful examinations were made of the face, trunk, arms, and legs with the view of determining whether any change of sensation was present. None could be determined except at one or two examinations she seemed to have on the postaxial border of the upper limb slight impairment of superficial touch, as tested by the von Frey hairs and cotton wool. No impairment of the senses of pain, heat and cold, or pressure was anywhere present. Some impairment of the sense of position was apparently present in the left hand, but this was very moderate and it was sometimes doubtful as to whether it was really present or whether the results were due to loss of power. Her fingers were used quite awkwardly, seemingly both from the impairment of power and of the muscular sense. This was particularly noticeable in observing the manner in which she used the index finger and thumb of the left hand as compared with the use of the right hand in manipulating objects. She recognized coins in the left hand, but some little difficulty was caused by the awkwardness of her movements. She had no positive loss in the recognition of objects. Diadokokinesis was present in an imperfect degree in the left hand, but not in the right.

A comparison of the results of the examination of the deep reflexes in the upper and lower extremities was instructive. The von Bechterew scapulo-humeral reflex was present on both sides, but more readily elicited and more marked on the right side. The coraco-brachial reflex was present on both sides, a little more marked on the right. The biceps tendon reflex was present on both sides, but distinctly less marked on the left than on the right. The triceps tendon reflex was present, but not very marked on either side. The muscle jerks were prompt on both sides. Comparing the results obtained in the left lower extremity with those in the left upper, a notable difference was evident. In the left lower extremity the deep reflexes were all extremely marked. The knee jerk was very much exaggerated, and a typical Babinski reflex was present. No patellar or foot clonus was present. In the right lower extremity the knee jerk was marked, but less than on the left. No foot clonus or Babinski reflex could be elicited. At some examinations no metatarso-phalangeal response occurred from plantar stimulation.

The tonicity of the left lower extremity was somewhat increased while that of each upper extremity and of the right lower extremity was about normal.

An electrical examination by Dr. A. R. Allen was made February 23, 1910. The various muscle groups reacted quickly to the faradic current. With the galvanic current the kathodal closing contraction was in all cases greater than the anodal closing contraction. The muscles tested were those of the shoulder girdle on the left, the left arm, anterior and posterior aspects, anterior and posterior aspects of the left forearm, and the intrinsic muscles of the left hand.

Dr. H. K. Pancoast reported the result of his Roentgen ray investigation as follows: "The skiagraphs show a shadow outside of the fourth and fifth cervical vertebræ on the left side. This shadow suggests osseous tissue and does not resemble sarcoma. The mass is small and lying in such a position that it might readily catch the posterior roots and press on them. The left halves of the fourth, fifth and sixth vertebral bodies seem a little denser than do the right halves."

The report is interesting as it apparently showed osseous rather than intradural disease, as was demonstrated by the operation. It shows that one may misinterpret somewhat the skiagraphic determinations in such a case. The misinterpretation however was not of such a character as to cause any doubt as to the propriety of surgical procedure. The shadow showing an apparent increased density of the bone, must in some way have been produced by the presence of the somewhat dense tumor,

tumor there may have been also some increased density of the bone itself which was not however determined. An endothelioma may by irritation cause some hypertrophy of overlying bone.

Dr. Charles H. Frazier's Notes of the Operation

A laminectomy was performed under ether anesthesia preceded by morphine and atropine. A semi-lunar skin flap was made and an incision made in this flap to the left of the median line. This was lifted up by means of a chisel, and the muscles and flesh were stripped from the underlying vertebræ. The spinous processes of the fourth cervical vertebra, the spine and lamina of the fifth cervical and a portion of the lamina of the sixth cervical vertebra were removed. There was very little hemorrhage. The dura was opened, and a small tumor about the size of a horse chestnut was located a little above the level of the fifth vertebra. The posterior root of the sixth cervical nerve ran over the mass, and it was impossible to say whether the anterior root ran through it or not. The posterior root of the fifth cervical nerve ran over the upper end of the mass. The tumor was very friable and was removed piecemeal by a curet and forceps. At times in removing the tumor there would be marked twitching of the left shoulder. The dura was closed and the skin and flesh put down after the introduction of a small cigarette drain.

The pathological report was as follows: The specimen is a portion of a tumor removed from the spinal cord. It is about the size of a pea and shows signs of hemorrhage and infiltration, the remaining portion of the tissue being white in color and soft in consistency.

Microscopically the growth is composed of cells which belong distinctly to the connective tissue group. They are mostly spindle shaped arranged in bundles which form interlacing whorls. In some places the growth is very loose, almost gelatinous in character. There is no fibrous envelope furnished by the processes of the tumor cells. The growth is irregularly canalized by blood vessels and stroma well marked. There is some fresh hemorrhage into the tissue. No mitotic figures are seen. The appearance corresponds to that of an endothelioma according to the views of Borst.—

The patient made a good surgical recovery, being able to sit up in about five days and was out of bed a day or two later. As soon as it was possible to make a neurological investigation without affecting the patient's surgical state, examinations were made. These showed improvement of the sensory, motor, trophic and other symptoms present before the operation, on the basis of which the diagnosis was made and the operative procedure advised. It will be only necessary here to give the results of a complete examination made nineteen days after the operation.

The patient stated that she felt unusually well as regards her general condition, and also that the pain had left the region of her left shoulder and left arm. She said that she was conscious of an ability to use her left leg and arm more easily and freely than before the operation. Her station was good, and she walked with freedom and a fair show of strength.

The left pupil was smaller than the right, as it had been ever since the operation. She had also a slight drooping of the left lid, and an inability to retract the lid as well as on the other side. When she looked straight ahead the left eye did not uncover quite as well as the right.

She used her arm on the left side more freely than before the operation. She was able to indent her fingers in her hand better than she did before the operation, but not as well as on the other side. There was a positive improvement as compared with the examination before the operation. The little finger no longer assumed the position of abduction and partial flexion which it did formerly; all the fingers including the little finger could be readily brought together and separated. Her grip was improved.

Tests for sensation in her arm showed no alteration in touch, pain or temperature. The sense of dulled perception which was formerly present on the back of the arm was now absent.

Since the operation the patient had complained of sensitiveness in the palm of the hand and on the palmar surface of the fingers, both subjectively and to pressure. Her left hand was more sensitive than the other when she washed it.

In the left upper extremity the deep reflexes—the tendon and muscle jerks—undoubtedly presented a different response from that exhibited before the operation. The von Bechterew scapular, coraco-brachial, triceps, biceps, wrist and finger jerks were all easily produced and quite as well marked as on the right side.

All movements of the lower extremity were preserved, showing little or no loss on the left side. No loss or impairment of sensation or hyperesthesia was present in the left lower extremity. The deep reflexes—knee jerk, quadriceps and ankle jerk, etc.—were all well marked and a Babinski response was present on the left; on the right all the deep reflexes were plus. No Babinski was present and no foot clonus on the right.[2]

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[3] Since the presentation of this paper at the meeting of the Association in May, another examination of the patient was made on June 9. The results of the examination were in most respects the same as of that made nineteen days after the operation. The arm and leg had retained all the improvement in power then noted. The pain in the shoulder and arm with which the patient had been so much distressed for many months, was absent.

This case presented the cardinal symptoms of a unilateral extramedullary tumor of the lower cervical region, in this designation including the first thoracic segment. The pain which was the first and always the most distressing symptom, indicated in the main the upper pole of the growth. The general impairment of power in both the upper and lower extremities, and the increased reflexes in the lower extremity, were due to compression of the pyramidal tracts of the left side. The absence of anesthesia, or at least its presence in only very slight degree, is probably to be explained by the fact that although two or more sensory roots may have been involved, the sensory supply from other roots replaced this loss by overlap. Besides the pain which the patient suffered in increasing degree as time went on, some cutaneous hyperesthesia was present in the upper arm, this also indicating the irritation of posterior roots.

The well-known Klumpke symptom complex consists of the ocular phenomena which were present in this case after the operation, plus more or less paralysis in the domain of the brachial plexus and especially in the distal portion of the upper extremity. This symptom complex can undoubtedly be produced by interference with the rami communicantes within the spinal canal. On the removal of the growth, the paresis previously present in the distribution of some of the component roots of the plexus, disappeared, leaving the unusual symptom complex here described.

The tumor proved to be an intradural one, probably growing from the inner surface of the dura. The differential diagnosis between an extradural and intradural but extramedullary, and an intramedullary tumor, has some interest in connection with this

The position of the fingers was practically normal. The patient however complained of some pain in the left index finger and forearm, this recurring every four or five days. When taking a bath she had some hyperesthesia to heat on the left half of the body, this passing away quickly. There was some impairment of the movements of the arm in trying to elevate it above the shoulder line or in moving it outward and upward. (This may be due to the cutting of the muscles of the back, chiefly of the trapezius, at the time of the operation.) The hand and finger movements were good with only slight impairment of the grip of the left hand. The reflexes of the upper extremities were all exaggerated and slightly more on the left. The lower extremities showed no weakness or sensory changes. The reflexes were normal on the right side but much exaggerated on the left, without foot clonus. The Babinski response was positive on the left. The eyes showed a slight degree of ptosis on the left though not so marked as when previously examined. The contraction of the left pupil was very slight.

case, and may be in any case a matter of some practical importance. It is certainly easier to discriminate between a tumor beginning in the medullary substance and one originating either from the inner or outer surface of the dura, than between the two forms of extramedullary growth, the extradural and the intradural. It is indeed practically impossible to make the latter distinction in some instances. In the first place, an intradural tumor, whether it originates from the dura or from the pia arachnoid, will have nerve root symptoms, posterior or anterior, similar to those of the extrameningeal growths, pain, hyperesthesia, impairment of sensation in root distributions when the posterior roots are implicated, and localized paralysis, atrophy, and vasomotor phenomena if the ventral roots are involved, also the results of interference with the vascular and lymphatic supply of the cord.

Later when the cord is compressed or invaded, true medullary symptoms begin to appear, those indicating for instance the involvement of the anterior or posterior horns and pyramidal or other tracts. The effects of compression of the pyramidal tract on one side will usually make their appearance early, these of course varying according to the location of the growth. If this be in the cervical region, some evidence of the pyramidal compression may be present in the arm, or such evidence may be masked by the more direct results of interference with the roots and cornual substance. The symptoms exhibited in the lower extremity will however be more or less purely pyramidal, showing themselves especially in some general loss of power in the limb and in exaggeration of the reflexes, both deep and superficial. All these points were well illustrated in our case.

The myosis, retraction of the eyeball and partial ptosis were post-operative symptoms probably due, as has been already intimated, to destruction within the spinal canal of the branches of communication from the cord to the sympathetic.

Case 2.—*Chronic spinal meningitis with subpial cyst; cyst evacuated; much improvement in voluntary movement of the limbs after the operation; cystitis; marked mental symptoms.*

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Although the opportunity was not offered by necropsy of determining the exact conditions within the spinal canal, the history and the results of operation in the following case would seem to indicate that it was probably one of chronic spinal meningitis or tumor, with a circumscribed subpial cyst. The patient was first seen by me in consultation with Dr. W. M. Hepburn, at Freehold, N. J. He was afterwards removed to the Hospital of the University of Pennsylvania on March 14, 1905, where a laminectomy was performed by Dr. Chas. H. Frazier, on March 20, 1905. The following is a somewhat condensed history of the case:

The first symptoms of the patient to which much attention was directed were in September, 1904, although prior to this he probably had had pain in the back and had not been quite like himself.

In September he went to bed with pain in the back and lower spine with some irritability of the bladder, that is he had to pass urine quite frequently and complained of pain in passing it. He was in bed for at least three weeks with this pain in the back and the accompanying symptoms. At this time he had no paralytic or spasmodic symptoms, simply the pain in the back.

Prior to this illness the man had a long history of what seemed to be perfect health. He was a man of excellent habits. He positively denied syphilis and had lived a very correct life.

He went along, the pain in the back not entirely leaving him, and after a time developed pain in the region of the left sciatic nerve. There was tenderness at the sciatic notch and also more or less pain and tenderness along the line of the nerve.

In the latter part of October, at a time when the weather was severe, there being a great wind storm, he had much increased pain in the back and leg, apparently as the result of taking some cold. He went to bed and was found to have a temperature of 103°. At this time he also complained much of the pain in the leg. This attack passed off in a week or ten days. On two or three occasions later, at intervals of about ten days, he had attacks not so severe, showing themselves with a higher temperature and increased pulse, accompanied by pain in the back and in the leg.

In the latter part of November he was taken with another attack without fever, in which he suffered pain over the region of the right kidney, and had also severe vomiting spells. He had one or two later attacks, and then these disappeared and never returned. He was much prostrated after these attacks, but gradually picked up until he was able to sit in a chair. Up to this time he had not been paralyzed in any way, although his pains had more or less continued.

In the latter part of December he was seen by Dr. Janeway, of New York, who discovered some anesthesia up to the level of the lower portion of the dorsal region, but nothing else, except the pain. Dr. Janeway thought that a tumor or caries might be developing.

About the middle of January the patient became paralyzed in both lower extremities, and for some days previous to this he had felt a weakness in his legs coming on. He could not stand up quite as well as he had done formerly.

Since this attack of paraplegia he had been in bed, helpless, at times suffering much pain, with inability to pass his urine part of the time.

A special examination was made by me just after his admission to the University Hospital, on March 14, 1905, the notes of which were recorded as follows:

As nearly as could be determined, the patient indicated that the pain was from about the mid-dorsal spine downward. As he described it, it seemed somewhat diffuse, although he indicated much pain in the lumbar and sacral regions. He complained that at times pains shot around the trunk towards the groin and also down the left leg.

He was totally paralyzed in both lower extremities, except that he could make slight movements of the whole leg, apparently by some of the trunkal muscles. Inspection showed that the left leg was larger than the right leg, especially below the knee, but he attributed this difference to a condition following typhoid fever some thirty years before. The left lower limb had probably enlarged as a result of a phlebitis following the typhoid attack.

Testing the reflexes in the right lower extremity, the knee jerk was found to be greatly exaggerated. Patellar clonus and foot clonus were very marked, the latter persistent; muscle jerks were also marked. The Babinski response was present.

In the left lower extremity the knee jerks were much increased. Patellar clonus was present. Persistent foot clonus could not be elicited, but it was imminent as shown by one or two oscillations. The Babinski response was present.

A not very elaborate examination for sensation showed that he was hypalgesic below a line about three inches above the umbilicus.

He was unable to pass his urine, requiring catheterization four times in twenty-four hours. In handling the patient it was noticed that he complained much of pain in the back and lower extremities. How much of this was psychic it was impossible to say. Sensation for heat and cold was not altered.

Electrical examination of the patient was made by Dr. Charles S. Potts, who reported as follows:

All muscles of both legs responded normally to the faradic current. The gluteal muscles responded well. Abdominal muscles responded well, but required a stronger current (about two inches of the nose of tube withdrawn) than did the muscles of the legs. The left side required a stronger current than the right. The muscles of the back in the lumbar region on the right side apparently did not respond to the faradic current, with the secondary coil switch on button, no. 2 tube completely withdrawn. On the left side there was a slight response with a current not quite so strong as this. The muscles in the upper part of the back responded well. The patient apparently knew when the muscles were contracting, and strong currents caused expressions of pain.

Examination of the eyes by Dr. G. E. de Schweinitz showed them to be absolutely normal.

On March 20, 1905, Dr. Frazier operated as follows:

When the dura was opened, in about the middle of the opening, at about the junction of the seventh and eighth vertebræ, a cyst protruded. It was 3.5 cm. in length. The upper and lower ends of this cyst were exposed; the wall of the cyst was formed by the pia; the fluid within the cyst was clear, and resembled cerebrospinal fluid. When a puncture was made in the cyst wall, the fluid escaped and the pia collapsed, and the cord then appeared to be normal. The cyst upon first appearance seemed to be gelatinous, but on further examination it was found not to be gelatinous.

The dura was closed with a continuous silk suture; the muscles and the skin with continuous catgut.

The patient remained in the hospital until June 14, 1905, when he was taken to his home. He died after some months but no necropsy was obtained.

He became more profoundly paralyzed, wasted and contractured. He continued to have severe cystitis. His mental condition was not good but was variable. He apparently died from exhaustion and secondary conditions.

A diary of his condition was kept at first from day to day, and later at short intervals. Briefly summarized the salient features of his condition after operation were as follows:

The pain at first was entirely relieved by the operation. Later at times he suffered considerable pain, especially in the abdominal region. Cystitis was present and was appropriately treated by irrigation, urotropine, etc. So much power returned in his lower extremities that he was able to draw the limbs up and down and to perform various gross movements with them. After a time voluntary movement returned, even in his toes. He had a variable temperature, pulse and respiration, this probably depending in part at least, upon the condition of his bladder. In a short time he developed rather active mental symptoms, suffering from more or less delirium, at times amounting almost to mania. Eventually a brace was used and he got up into a chair, but his mental and general condition did not greatly improve.

The appearance presented in this case when the dura was opened was almost identical with that observed in the case recorded by Spiller, Musser and Martin.[3] The result of the surgical interference was however, not as fortunate, but it will be remembered in the case recorded by these observers that the symptoms were much less pronounced. In their case the history had extended over several years, although the most important manifestations began two years before operation. The main feature for a long time was pain of a severe type, present in the left lower extremity and back, and at times in the extremity of the other side. As time advanced the pain increased and ramified to some extent, but always remained most marked in its early locations. The deep and superficial reflexes in the lower extremities in this case were at first much exaggerated but later changed, diminishing in the case of Achilles jerk on one side, and both knee jerks disappearing. Paralysis, contracture and atrophy never occurred, although weakness was present in the left leg. Anesthesias were not present but the patient complained of a pronounced paresthesia in the left foot. The bladder and rectum were not involved. The result of the operation by Dr. Martin was the relief of a large part of the pain suffered by the patient, this relief continuing up to the time of reporting the case several months after operation.

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[3] Spiller, Wm. G., Musser, J. H. and Martin, E., A Case of Intradural Spinal Cyst with Operation and Recovery, University of Pennsylvania Medical Bulletin, March and April, 1903.

Prior to the publication of the case of Spiller, Musser and Martin, two cases of spinal cyst had been recorded by H. Schlesinger,[4] in his monographs on tumors of the cord and vertebræ. In only one of these cases had a clinical study been made which could be referred to the lesion found on necropsy. The other cyst was discovered accidentally in a necropsy in a case of sclerosis of the brain and the spinal cord.

The first of these cases briefly summarized was that of a man thirty-six years old, the duration of whose disease was nine years. The first evidences of affection were pain in the neck and paresis in both legs. The symptoms exhibited frequent exacerbations and remissions and other vacillations. At times there was detrusor paralysis. After nine years duration a girdle sensation developed, with hypesthesia of the legs and slight bladder disturbance. Death occurred from an intercurrent disease. The circular or oval cyst, 3 cm. long was below the cervical enlargement and filled with clear fluid.

The case here recorded bore in some of its early features, a similarity to that of Spiller, Musser and Martin, especially in the presence of severe pain localized in the back and lower extremities, and in the exaggeration of the reflexes. Later, however, as will be recalled, atrophy, contracture and paralysis, with serious impairment of the functions of the bladder occurred.

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[4] Schlessinger, H., Beiträge zur Klinik der Rückenmarks- und Wirbeltumoren, 1898.

Bliss[5] has recorded two cases of cysts within the spinal canal. The case recorded in the Journal of the American Medical Association had much in common with the case here reported, being more similar to it than that of Spiller, Musser and Martin. In the case of Bliss there were anesthesia, paralysis, contracture, etc. The result of the operation was very satisfactory in the relief of pain, the return of sensation and power, and the improvement in the reflexes.

Bliss[6] has published another article briefly reviewing the subject of circumscribed serous spinal meningitis and especially calling attention to the cases of so-called cysts associated with chronic spinal meningitis, in which list, as I have already indicated, my case would probably fall.

Krause[7] has recorded three cases in which tumors were diagnosticated but cysts discovered at the operation. In two cases the fibrous overgrowths extended even into the cord substance. In two cases there was stasis of the fluid above the cysts so that a very marked variation in the symptoms occurred and the upper border of the disease was very hard to fix.

The subjects of chronic diffuse spinal meningitis with more or less generalized effusion and of circumscribed serous meningitis are of great importance in considering the question of tumors and cysts of the spinal cord, and demand our attention especially in discussing case two. Some of the first observations were made regarding this subject in this country, as Sir Victor Horsley[8] states, by Horatio C. Wood, of Philadelphia, in a contribution on syphilitic spinal meningitis, and other forms of syphilis of the nervous system. As regards the question of circumscribed serous meningitis, the first observation in this country, as also indicated by Horsley, was made in a paper by Drs. Spiller, Musser and Martin the one describing the case to which reference has just been made. At necropsy I have seen several cases of the sort described by Horsley, in which a diffuse pachymeningitis or a condition of hypertrophic pachymeningitis with several foci of great thickening were found.

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[5] Bliss, M. A., Cysts Within the Spinal Canal, Journal of the American Medical Association, Vol. 52, No. 11, March 13, 1909.

[6] Bliss, M. A., Circumscribed Serous Spinal Meningitis, Inter-state Medical Journal, Vol. XVI, No. 5, May, 1909.

[7] Krause, F., Ueber Schwartenbildung am Ruckenmark (Meningitis fibrosa chronicalis), Die Therapie der Gegenwart., Vol. XI, No. 12, December, 1909.

[8] Horsley, Sir Victor, Chronic Spinal Meningitis, The British Medical Journal, Vol. I, Feb. 27, 1909.

In the cases of chronic spinal meningitis to which Horsley refers, the excessive cerebrospinal fluid present is not always, or perhaps frequently, in a strict sense, circumscribed. It extends more or less up and down the cord, with a place of greater accumulation at some point. This place of special distension or enlargement may serve as a guide to the site and upper limit of operation.

It is a remarkable fact that Horsley has operated on no less than twenty-one cases of chronic spinal meningitis with more or less serous effusion, and has been able to bring about either a cure or great improvement in a number of cases. He has also demonstrated the real pathology in these cases by at least two necropsies with microscopical examination. In one of these an operation had been performed; in the other the true nature of the case was discovered, or at least proved, at the necropsy. The membranes in these cases were greatly thickened. It might be said in passing that the areas of thickening in such cases may differ greatly at different heights.

Some of the diagnostic points between these cases of diffuse or of circumscribed serous meningitis and spinal tumor are, as pointed out by Horsley: (1) In tumor the pain is apt to begin and to be more or less limited to a single nerve area, whereas in chronic meningitis a number of nerve distributions may be affected. The whole column may take more or less part in the pain. (2) Not only does this diffusion of symptoms and signs hold good with regard to pain, but there may be also a larger diffusion of such symptoms as paralysis, atrophy, cutaneous eruptions, etc. (3) The case progresses somewhat differently from that of a tumor.

Oppenheim[9] regards as characteristic of meningitis as compared with tumor only the slow progress of the symptoms and the tendency to remission, and claims that even with these present, tumor and meningitis may be confused.

[9] Oppenheim, H., Diagnose und Behandlung der Geschwülste innerhalb des Wirbelkanals, Deutsch. med. Woch., No. 44, Nov. 4, 1909.

Cases of tumor, as well as the cases described by Horsley, must be differentiated from cases of cysts of a strictly circumscribed character like one recently presented by Weisenburg[10] at a meeting of the Philadelphia Neurological Society. In this case the affection was supposed during life to be a tumor, but a circumscribed serous cyst was found and the patient was completely cured by operation. The point made by Weisenburg in the presentation of this case can probably be maintained only in a limited number of cases, namely that a differentiation between a spinal cord tumor and a cyst can sometimes be decided by vacillation in the focal symptoms. In his case, for instance, the sensory symptoms present on one day or at one time, representing one half of the cord, at the next examination were found to be indicative of involvement of both sides of the cord.

In the case of a cyst of the spinal cord, as in one of the brain, some of the symptoms show an unusual variability. In a brain cyst, for instance, which is associated, as is not infrequently the case, with tumor, the vacillation in some of the symptoms leads to doubt as to diagnosis, and the same is true, although to a less extent, of lesions purely spinal. Impairment of movement, and changes, impairment or perversion of sensibility, and even the state of the reflexes may differ from time to time, probably because of variations in pressure according to the condition of the cyst at different times. Similarly the level of a spinal lesion will seem to vary in an unusual way as indicated by the differences in such manifestations as hyperesthesia or anesthesia, paresis or reflex disturbances, and this apparently in accord with the relative filling and emptying of the cyst. The amount of fluid in these cysts evidently varies at different times. This may depend upon several circumstances, variations in the conditions of the lesion which induce the secretion, absorption under the influence of treatment, and variations in the position of the patient.

Care must be taken in drawing conclusions from variable conditions of this sort in determining whether the cyst, if it is associated with a tumor, is producing these variations from above or below the level of the mass. In the case of a purely cystic formation the variations will probably be in all directions. Such variations however, will chiefly take place where the condition is one of stasis associated with tumor or meningitis.

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[10] Weisenburg, T. H., Idiopathic Circumscribed Spinal Serous Meningitis with the Report of a Successful Operative Case, The American Journal of Medical Sciences, 1910.

In discussing the question of vacillation of symptoms in different forms of lesion under consideration, Oppenheim says that the most important sign in the meningeal growth is the constancy of the upper pole symptoms, while in the intramedullary growth there is a tendency to a meningeal spinal extension with a change upward in the level of the symptoms.

As a rule a serous or other cyst, if unassociated with a tumor or meningitis, will not cause the same degree of pain which is produced by a tumor involving membranes of the cord, especially the posterior nerve roots, but such a rule is not without exception.

In the case reported by Spiller, Musser and Martin, pain in the lower extremity and also in the back was a prominent symptom, and was relieved by an operation which resulted in the discharge of what appeared to be a circumscribed serous cyst.

With regard to the subjects under discussion Oppenheim has probably written more fully and lucidly than any other contemporary neurologist. With regard to circumscribed serous meningitis (meningitis serosa spinalis circumscripta adhesiva), he says that in this there is "A fibrous inflammatory process forming adhesions with the arachnoid. These spaces then become filled by an increase of fluid. One may find several cysts formed, or only one cyst with stasis above the cyst, but with a free circulation of the fluid upwards. This process may occur alone or secondary to a tumor or vertebral disease."

If upon operating for either a tumor or a serous condition, the dura is found greatly stretched and non-pulsating, and upon cutting it, it is found that the liquid gushes out under considerable pressure and in large amount, the meninges seeming however otherwise normal, meningitis serosa is not the only disease present, but the condition is a fluid stasis above or below the tumor. By sounding or further laminectomies the tumor will be found. If by sounding bounding walls are found it is a serosa circumscripta.

Serosa circumscripta may even cause a flattening of the cord at the level of the cyst. There may be however, an effusion in the membranes with the formation of a cyst above a tumor caused by edema of the arachnoid.

In the case of circumscribed cyst here described, the probabilities are great that this formation was associated either with a tumor or with a meningitis situated mainly below the site of the cyst. This is indicated by the merely temporary relief of some of the important symptoms, and by the subsequent course of the disease to a fatal termination.

The Indications for the Antisyphilitic Treatment of Tabes Dorsalis.[1]

By JOHN H. W. RHEIN, M. D.,
Neurologist to the Howard Hospital, Philadelphia.

(Read in the Section on Medicine, Medical Society of the State of Pennsylvania, Philadelphia Session, September 29, 1909.)

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The value of the antisyphilitic treatment in tabes is still a matter of dispute. While much has been written on the subject, there exists among equally reliable authorities no unanimity as to the value of this treatment. Recently I have made some studies which incidentally bear on this subject, and while the observations add little that is not already known, I believe they are worthy of brief mention.

It is pretty generally conceded that syphilis is the cause of tabes, although just how it operates in causing the pathological conditions found in this disease is yet a matter of controversy.

Whether syphilis causes the degeneration in the spinal cord by means of infection through a posterior lymphatic system, a theory advanced recently by Marie and Guillain,[2] or is due to a strangulation of the posterior roots by reason of the specific meningitis present (Redlich[3] and Obersteiner[4]), or to a toxic action of parasyphilitic origin, or to syphilitic infiltration of the radicular nerves, as claimed by Nageotte,[5] matters little in our present discussion. It is sufficient to admit merely what I believe is generally conceded, that the cause of the degeneration of the sensory neuron is syphilis, whether it be direct or indirect.

Neither antisyphilitic, nor any other form of treatment, could possibly affect favorably the tracts which are already the seats of degeneration. No one can hope to restore degenerated nerve fibers of the spinal cord by means of any treatment yet known. The only results, therefore, that one could expect from an antisyphilitic treatment would be the prevention of the extension of the tabetic process by removing the primary cause of the degeneration, whether it be due to meningitis around the posterior roots, to the syphilitic infiltration of the radicular nerves, or to a syphilitic lesion of the posterior lymphatic system, or what not.

The antisyphilitic treatment, therefore, can not possibly be considered to be a curative treatment, as has been claimed by some, for obvious reasons. Nor can it be looked upon as of any value in the neutralization of a syphilitic toxin, and therefore of no value if tabes is a purely parasyphilitic disease, except to act upon the source of the parasyphilitic toxin and prevent any new formation of such toxins.

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[1] From the Department of Neurology and the Laboratory of Neuropathology at the University of Pennsylvania.

[2] *Revue Neurolog.*, 1903, Vol. 11, p. 49.

[3] *Deut. Path. der Tabisch. Hinterstrangerkrank.*, 1897.

[4] *Nervösen Centralorg.*, 1901, p: 321.

[5] Cornil and Ranvier, *Manual d'Hist. Path.*, 1907, No. 3. p. 316.

While there exists a number of authorities who are unqualified in their opinion as to the value of the antisyphilitic treatment, there are others who have failed to observe any benefit whatever from its use. Among those who have been enthusiastic as to its value are Fournier,[6] Erb, Vaudey,[7] Dieulafoy,[8] Gaucher,[8] and others.

Paviot[9] believed that all the evidence was in favor of admitting a meningeal inflammation as the cause of the degeneration in the posterior roots, and claimed that this was the reason for persisting in the mixed antisyphilitic treatment, rather to prevent the extension of the disease and to effect an arrest in its evolution, than to hope to recuperate the degenerated fibers.

Cauvy[10] believed it of great value at the onset during the evolution of the disease. He advises it also in cases which are apparently stationary, to prevent further progress.

In Faure's[11] opinion certain syphilitic cases of tabes vigorously treated at the onset of a case in rapid evolution by mercurial treatment are sometimes cured. It should always be tried in the early stages.

Vaudey[7] quotes cases treated by the antisyphilitic treatment in which the results show great improvement, although he admits that he never saw the knee-jerks return, or any disappearance of the fulgurant pains. He advises intense mercurial treatment whether there is specific history or not.

Oltramare[12] cites good results in one case, and Dinkler,[13] as a result of the treatment of seventy-one cases, concluded that no unfavorable effects were seen due to this treatment, and in the majority of cases improvement was shown.

M. A. Starr[14] advises the treatment in cases where there is a history of previous infection, or wherever its existence is suspected, and in his experience it has proved of benefit in a considerable number of cases. He calls attention to the fact that the use of mercury hastens the process of optic nerve atrophy, and therefore its use is contraindicated in those cases in which the optic atrophy is the initial symptom.

On the other hand many authorities are skeptical of the beneficial effect of this treatment. Charcot[15] has seen a negative action from it, and Oppenheim[16] has never seen any good results.

Marie,[17] while recognizing the syphilitic origin of tabes, claims that the antisyphilitic treatment has no effect apparently, and believes it is harmful when cachexia is present.

Senator, Redlich and Eulenberg[18] believe that the effect of this treatment is limited, and Ballet,[19] in an interview with Donadieu-Lavit, stated that he ordered mercury in tabes, but he did not believe in it, and that he had never seen a cure by mercury.

Gowers[20] believed that the antisyphilitic treatment seldom did good, and sometimes was harmful in cases of slow development several years after the primary infection. He believed that energetic mercurial treatment is harmful in the late cases with degeneration that follows syphilis, believing that the depressing effect of such treatment increased the tendency to degeneration and hastened the process. Dana[21] believes that in the majority of cases the treatment is not useful, and may actually do harm, and in the opinion of Sarbo[22] the results of the treatment are uncertain, and he advises its use only in the early stages.

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[6] Vaudey.
[7] *Marseilles Med.*, 1904, Vol. 22, p. 193.
[8] Rauzier.
[9] *Lyon Med.*, 1905, p. 913.
[10] *Annal. des Mal. Vener.*, 1903, p. 352.
[11] *Gaz. des Hop.*, 1904, 77, p. 669, and London *Lancet*, 1904, Vol. 1, p. 717.
[12] *Jour. des Mal. Cut. et Syph.*, Paris, 1904, 169.
[13] *Berlin. klin. Woch.*, 1893, Vol. 39, p. 347.
[14] Organic Nerv. Dis., 1903, p. 324.
[15] *Montp. Med.*, 1896, p. 641.
[16] Diseases of the Nerv. Syst., 1904, 2d Am. edition, p. 147.
[17] Lecture on Dis. of the Spinal Cord, 1895, p. 315.
[18] Nonne, Syph. und Nerv. Sys., 1902, p. 325.
[19] Donadieu-Lavit, *Montp. Med.*, 1904, Vol. 18, p. 421.
[20] Dis. of the Nerv. Syst., Vol. 1, p. 436.
[21] Text-book of Nerv. Dis., 1897, p. 292.
[22] *Wien. klin. Woch.*, 1907, p. 1312.

The object of this paper is not entirely to review the literature of this subject, but particularly to put on record the results of an examination of the cerebrospinal meninges in fourteen cases of tabes dorsalis, and the spinal meninges in two additional cases. These sixteen cases all showed more or less round-cell infiltration of the pia. This round-cell infiltration was not typically specific in all cases, however, although only differing in degree from the round-cell infiltration of specific origin. A typically specific meningitis was found in five cases. It was impossible in these cases to make any deduction from any data that could be gained from a study of the treatment employed in these cases.

In view of the findings in my cases, *i. e.* the presence of a meningitis in all, with a typically specific meningitis in five, I believe I am warranted in the conclusion that an antisyphilitic treatment should be tried in all cases of tabes, and that the persistence and intensity of the treatment should be regulated by the progress of the case. I do not believe it is necessary to be governed by whether or not there is a positive specific history or whether there are other syphilitic symptoms present, although in my opinion a history of incomplete treatment or of symptoms indicating other syphilitic manifestations only serves to emphasize the importance of this treatment.

I advise the antisyphilitic treatment in tabes in spite of the fact that I have never been convinced that the treatment in my hands has been of much, if any, value. But it is my belief that the antisyphilitic treatment should have a favorable influence upon the meningitis which, I believe, it is proper to conclude is present in most of the cases.

While such a treatment may not have any direct bearing upon the progress of tabes, it certainly can not but favorably affect the meningitis which in many, if not all, of the cases is syphilitic in origin; and if this meningitis is not the cause of the degeneration in the posterior columns, as is claimed by some, and which I believe is doubtful, yet it is conceivable that the presence of such a meningitis can not but unfavorably influence the progress of the tabetic process.

The treatment should not, however, be prosecuted when cachexia, due to chronic liver, kidney, lung or other diseases, is present, and where the administration of the iodids and mercury increases autoinfections of a gastrointestinal type, or where optic atrophy is an early symptom.

I agree with Troelich[23] and others who advise the employment of this treatment in those cases of tabes which are associated with florid syphilitic processes.

The cases of tabes dorsalis examined, consisted of the available material of the laboratory of neuropathology at the University of Pennsylvania, the opportunity of studying which having been afforded me by the courtesy of Professor William G. Spiller.

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[23] *Deut. Aerzte-Zeit. Berl.*, 1907, p. 97.

MENTAL SYMPTOMS OF SYPHILIS*

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While it is not hoped in this communication to offer anything original upon this question, the subject, having been recently brought to my notice by a few cases which have come under my observation, was suggested as an interesting one for discussion before this society.

For half a century in the general literature a number of communications have been made bearing on the subject, among the earliest of which are those by Hildebrand, in 1859, and Wille in 1871-72. But Fournier[1] in 1879 was perhaps the first to consider this subject at any great length. To him we are indebted for the first descriptions of the so-called pseudo-general paralysis of syphilitic origin, though at the same time he described other forms of mental disease which he attributed to syphilis.

It is to be especially noted in reviewing the literature of the subject that many forms—it might almost be said, all known forms—of insanity have been described as being caused by syphilis. Pandy[2] recently made the statement that in asylums lues was found to be the cause not only of general paralysis but typical melancholia, mania, amentia, paranoia, pseudo-delirium tremens, idiocy and epilepsy, and Finckh[3] believed that all forms of psychic symptoms could be attributed to syphilis.

Syphilis may act in several ways. It may cause a moral shock, giving rise to melancholic hypochrondriacal conditions and hysteric and neurasthenic states which have no direct relation with the syphilitic poison; or the toxins and the cachexia, the result of the general syphilitic process, may give rise to psychopathic states; or specific processes in the brain and its membranes may give rise to a train of symptoms called pseudo-general paralysis; or, finally, psychoses may be of a parasyphilitic origin. The psychoses that develop as the result of the moral shock to the nervous system and the worry and anxiety entailed by having acquired syphilis has really nothing to do with the subject in hand.

The toxins of syphilis give rise, according to many authorities, to the development of melancholia, mania (Jolly[4]), manic-depressive insanity (Bianchi[5]) and neurasthenic and hysteric complexes. It is a question, however, whether syphilis can be said to cause a true independent symptom complex. Indeed, Angiolella and Goldi[6], Finckh and others have recently denied that the psychoses of syphilis have an independent clinical entity.

It is also a question with some authorities whether the syphilitic process causes mental symptoms at all. Krause[7] and Marchand[8], for example, believed that the specific psychoses were the outcome of disturbances of nutrition of the brain cortex caused by syphilitic toxins and, according to Salager[9], hallucinations (Verwirrtheitzustand) result from the cachexia of syphilis, due to the lesions of the kidney, liver and arteries, rather than a specific lesion of the brain and its membranes.

While syphilis appears to be an exciting cause at least in a variety of psychopathic states, there is nothing characteristic in these cases except perhaps a tendency to mental deterioration or dementia. Cases of paranoia of syphilitic origin have, however, been reported by Henneberg[10] in which specific meningo-myelitis was present at the autopsy, and by Kowalewsky[11], Jolly, Krause and others, though instances of this kind must be rare, and the question arises whether or not such an association is not accidental.

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*Read before the Philadelphia Psychiatric Society, May 13th, 1910.

An unusual form of mental disease of specific origin is the Korsakows symptom-complex due to syphilis, cases of which have been cited by Rosenheld[12] and Jolly. Cases of insanity of irregular type have been described, examples of which are those of Walker, Diller and Burns[13], Hotchkis[14] and Mallison[15].

In the case reported by Walker, Diller and Burns, hallucinations of sight and hearing, stuporous delirium and advancing mania, associated with optic neuritis, bulbar symptoms and monoplegia constituted the chief symptoms, while post-mortem, round cell infiltration of the cortex and vascular disease were demonstrated.

In Mallison's case there were mental confusion, violence and incoherence, the patient was grandiose, the pupils were immobile, the knee jerks were increased, and nodes on the tibia were present. At the autopsy syphilitic brain disease was found. Hotchkis described a case in which incoherence, excitement and defective memory followed a cerebral hemorrhage, having recovered a year previously from a psychosis consisting chiefly of hallucinations, delusions, incoherence and periods of violence.

A few instances of insanity occurring during the florid stage of syphilis have been reported. Capuccio's[16] case was one in which there occurred during the secondary stage of syphilis, fever with visual hallucinations. O'Donovan[17] described a case of acute transitory mania, and in a case cited by Schwab[18] there was motor restlessness, complete amnesia, tremor of the hands, then finally the picture of a typical dement. In Berkley's[19] case of acute insanity in the florid stage of syphilis the patient presented the symptoms of violent mania, followed by stupor without delusions or hallucinations, with gradual recovery.

The so-called "pseudo-paralysie generale" (Fournier) has been variously described as progressive dementia (Krafft-Ebing[20]), post-syphilitic dementia (Ziehen[21] and Binswanger[22]), organic syphilitic psychosis (Mendel[23]), syphilitic dementia (Bianchi), dementia paralytica syphilitica primaria (Kowalewsky), leutic pseudo-paralysis (Kraepelin[24]), and finally, as Mott[25] prefers, syphilitic brain disease.

This symptom complex which deserves to be recognized as a clinical entity I believe, consists of psychic disorder added to somatic disturbances. It is a group of symptoms which results from the invasion of the brain and its membrane by the syphilitic process, consisting of round cell infiltration and gummatous deposits in the membranes and substance of the brain itself, or specific arterial disease leading to softening and malnutrition of the brain substance. The variation of the process and its distribution explains perhaps the variability of the symptoms which every one recognizes as peculiar not only to syphilitic psychosis but to syphilitic disease of other organs.

The psychical phenomena are varied, and only characteristic in that there is a tendency to dementia. The deep alteration of the intellectual activity has been emphasized by Kowalewsky, Krafft-Ebing, Erlenmeyer[26], Ziehen and others. Ziehen describes psychic states associated with physical symptoms of brain syphilis, consisting of headache, vomiting, vertigo, convulsive disturbances, paralysis of pupils and other cranial nerve palsies, choked disc, hemiplegia and the like. The psychic phenomena consist of grandiose ideas, hallucinatory paranoid states, stuporous conditions, hallucinations alternating with stupor, or simple intellectual defect.

Krafft-Ebing described the mental symptoms as consisting of a progressive dementia in which appear primordial delusions of persecution and of grandeur, hallucinatory delirium, profound somnolence and dreamy states, and violent mania ranging to acute delirium. The fundamental psychic weakness, the marked disturbance of consciousness, sudden occurrences and subsidence of symptoms are more or less characteristic features of this symptom complex.

According to Mendel, the psychical symptoms consist of loss of memory and hallucinatory delirium; hypochondriacal melancholia or mania alternating with depression, and rarely a paranoid mania. These psychical phenomena are associated with somatic symptoms consisting of headache, convulsions, hemiplegia and disturbance of speech, such as dysarthrias and dysphasias. The psychical symptoms of syphilitic mania, according to Fournier, consist of simple excitation, exaltation, incoherence, excitation with mental hebetude, delirium, maniacal delirium, etc.

It is characteristic of this disease that the symptoms may be materially ameliorated by anti-syphilitic treatment and that there is a variability in the symptoms with fundamental psychic weakness and marked disturbance of consciousness. It differs from true paresis in that in the former there is rarely or never, tremor of the lips or tongue, the typical speech disturbances of paresis are lacking, the physical state is impoverished, and while there is a progressive tendency, the course of the disease is characterized by sudden insults and variability of symptoms, while in true paresis the disease is insidious and progressive.

As demonstrated by Plaut[27], the Wasserman reaction as well as the study of the cerebro-spinal fluid for lymphocytosis should prove to be of great value in the differential diagnosis.

The keynote of this whole study seems to me to be the recognition of the value and importance of instituting at an early period the anti-syphilitic treatment. The diagnosis of these cases should be easy when we recognize in this disease an irregular psychical state added to characteristic somatic symptoms of tertiary syphilis.

Case I.—The following is a case illustrating a psychosis occurring a year after the florid stage of syphilis. A year previous to the onset of the psychic symptoms the patient, aged 21, had contracted syphilis. After returning to this city from a trip to Washington, D. C., on March 4, 1905, he suffered from insomnia, general tremor, became dilatory in his reading and wandered about the house aimlessly by day and night. Three weeks later he began to have difficulty in emptying the bladder. On April 7, in the evening, he left his room in a hotel to go to the toilet and wandered into the wrong room, reappearing in his own room at 5 o'clock in the morning, and said, upon being questioned, that he had been absent for five minutes only. He finally required catheterization at this time. Two days after this incident he had a general convulsion with unconsciousness, foaming at the mouth and biting the tongue. A similar attack followed in a week and a third attack two days later.

Upon examination of the patient on April 21, 1905, which was six weeks after the onset of the symptoms, his condition was as follows: There was partial double ptosis which could be partially overcome on voluntary effort. The eyelids could be closed but efforts to open them on command were only partially successful. When he attempted to focus upon the finger of the examiner held in front of him a weakness of the external rectus of the left eye was observed. The tongue was protruded to the right tremulously. No facial palsy was present and there was apparently no paralysis of the arms and legs. There was incontinence of feces and paralytic retention of urine. He had two bed sores on the buttocks and one on the right heel. There was no apparent loss of sensation. The knee jerks were increased; the arm jerks were present, and there was no clonus. The Babinski phenomenon was present on both sides. There was present general choreiform movements and the general musculature was tremulous. Carphalogia and subsultus tendinum were present. He was not able to answer questions clearly, he was dull and it was difficult for him to concentrate; in fact, later he became semi-stuporous.

There was associated with this condition a continuous temperature for about three weeks. At the end of a few weeks, general improvement ensued and finally the patient became entirely well.

Case II.—The second case is one which deserves but a brief mention and illustrates the gradual dementia that accompanies tertiary syphilis. The case was that of a man who developed, at the age of 35, the symptoms of general cerebro-spinal syphilis, consisting at first of spastic paraplegia and later apoplectiform attacks, transient third nerve palsies and hemiplegias, the disease lasting nine years and finally terminating in death. In the last four years of his life there was a gradual mental failure. There was distinct change in his character, he lost his natural sense of modesty, he was silly, flippant, frivolous and indifferent to his condition. There was distinct loss of memory and in fact the general mental picture was that of a progressive simple dementia.

Case III.—The following case represents in my experience the usual type of syphilitic psychosis, except that the psychical phenomena preceded the somatic symptoms which, I take it, is the reverse of the usual mode of onset.

E. W., a colored woman, age 42. Family

and previous history unobtainable. She had been apparently in perfect health until March 8, 1910, performing the duties of a cook, without showing any symptoms that had been recognized. On the morning of March 8th, she was found preparing breakfast for her mistress on a range in which the fire had gone out. She used the wrong dishes in preparing the food moreover, and did not seem to recognize that she had been making mistakes. After taking her to her home her son informed me that she would arise in the morning at a very early hour, about 3 o'clock, put on her corsets and shoes and, believing that she was properly dressed and that it was daylight, attempted to go from her home to her former place of employment. Her talk was somewhat incoherent, usually upon subjects pertaining to her occupation—that of a cook. At the end of a week she was admitted to the Howard Hospital, where her condition was as follows:

Physical examination was practically negative except that the knee jerks were increased and there was on the right knee a multiple eruption which was diagnosed by Dr. Knowles as the skin eruption of tertiary syphilis. There was also just over the tibia on the right side, a gummatous node. The gait was rather feeble but not characteristic. She passed the contents of the bowels and the bladder incontinently, probably due to her mental condition. There was no change in sensation to the prick of a pin point. The tongue was protruded straight and no facial palsies or palsies elsewhere were discovered at this examination. Examination of her mental condition showed that she was disoriented as to time and place, had apparently lost her memory for recent events, failing to recognize her physician whom she had known for some months. She spoke only when addressed, was dirty, somewhat resistive—in short, presented the mental picture of partial dementia.

She was admitted to the Philadelphia General Hospital March 25, 1910, and to the Philadelphia Hospital for the Insane, March 30, 1910. She made the statement, upon admission to the hospital, that the Lord spoke to her, and said He would forgive her sins, and that she saw the Lord, and that He looked like a natural man, had a mustache, but no beard, and was medium sized. She knew the Lord because He sat and talked to her. Says she saw certain people, now dead, known to her long ago, who visited her and talked to her.

Examination made by myself, April 25, 1910, resulted as follows: When told to put out her tongue, she understood what was meant but was apparently unable to protrude the tongue but a short distance beyond the teeth. The speech was thick, indistinct and unintelligible. She was mentally very dull and indifferent. She was completely disoriented, believing she was at the house of a former master, on Gun Powder road, and that she was boarding at that place. No hallucinations or delusions could be developed and the mental condition appears to be one of dementia and confusion, with memory for old events fairly well preserved, but for recent events entirely lost.

She could not support herself on her feet, due to weakness of the left leg, and when attempting to stand developed a general tremor, almost convulsive-like, contractions of both arms and legs occurring. There is apparently no power in the left arm, and the left leg was flaccid, and could be moved only slightly. The right arm and leg were distinctly spastic but could not be moved on command. Both knee-jerks were increased, but more so on the right. Her bladder and rectum were incontinent. The left angle of the mouth drooped when efforts at speech were made. The eyeballs moved in all directions, following the finger. She drew the right hand promptly away from the pin point but did not move the left hand when it was pricked. She felt the pin prick apparently in both legs. She had to be fed. The paralytic condition was of three days' duration and was evidently of insidious onset. The pupils were equal and responded normally. There was no heart murmur and the lungs were clear. Until a few days prior to my examination she was very restless, at times requiring slight restraint to keep her in bed.

She died on May 10, 1910, and the brain and spinal cord were removed and placed in formalin solution.

A small nodule was seen on the basal surface of the right frontal lobe in front of the optic chiasm, which was attached to the brain substance but principally implicated the pia. A small tumor was observed on the right side in the outer and inferior portion of the middle of the Pons. There was also marked thickening of the pia, especially over the base of the brain, but also over the convexity.

Sections were made from the tumors and from the paracentral and other regions and stained with hemalum and acid fuchsin. The tumors proved to be gummata, and there was marked specific round cell infiltration of the pia which in places involved the cortex. The vessels of the pia were thickened and in some there was proliferation of the intima. There was in the cortex from the paracentral and precentral regions some perivascular distention and in the cortex of the prefrontal region areas of intense round cell infiltration were seen.

This case was one in which the autopsy revealed the characteristic changes of tertiary syphilis and the symptoms were without doubt due to the pathological conditions which were found in the brain and its coverings.

Case IV.—The next case is one which is atypical and may well be considered a doubtful case of true syphilitic psychoses. The character of the life led by this patient may have as important a bearing upon the causation of the mental symptoms as the syphilis itself.

M. R., aged 29 years, female, family history negative. Mother stated that at the age of 16, before her marriage, she contracted syphilis, that after being married, at the end of three weeks she infected her husband, who left her at once. During the past seven years she has been living with another man and was not married to him. The present symptoms began two years ago.

On March 23, 1910, on admission, her condition was about the same as when I examined her on April 25, 1910. The chest, back, arms, hips and legs were covered with a coppery eruption, crescentic in shape, which was diagnosed as a tertiary syphilitic skin eruption. There was no facial palsy. The tongue was protruded straight and in the median line. The eyeballs moved freely in all directions following the finger of the examiner. The pupils contracted to convergence, in accommodation and to light. The grasp on both sides was feeble, but there was no palsy of arms or legs. There developed upon voluntary effort a fine, fibrillary tremor of both arms.

The knee jerks were equal and very much increased. The achilles jerk was prompt and equal on both sides. There was an ankle clonus on the left side which was readily exhausted. Stroking the toes of the feet caused flexion of all the toes of both feet. There was apparently general slight hyperesthesia. Grasping the arms or legs, or tapping the head, called forth a complaint of pain. There was no loss of sensation to the pin point. The station was good and there was general muscular enfeeblement. There was incontinence of urine, probably due to the mental state. She fed herself. The skin eruption was fading and had almost disappeared at this examination. During the physical examination patient talked continuously in a rather rambling manner about her physical condition.

The mental condition may be described as showing partial dementia. There were no hallucinations or delusions; the questions were answered promptly, showing fairly good memory for past events, but there was a tendency to drift from the subject, and her remarks were entirely irrelevant. She was not depressed or elated. She complained of headache when asked, but did not volunteer any complaint of headache. Her expression was rather vacant.

Case V.—The following case is an illustration of syphilitic insanity showing delusions and dementia with some excitement.

W. P., age 34, family history negative, married, clerk by occupation. He had an initial lesion in May, 1907, a secondary rash in July, and in December syphilitic sore throat and destructive ulceration of his palate.

On admission November 11, 1908, there was general lymphangitis. Shortly after admission he began to act queerly, attempting to leave the institution without permission. This was followed by general confusion and some delusions. He said that his mother had been changed to a colored man, and was about to be killed. He believed that the colored man in the next bed was his mother, and tried to get in bed with him. He stated that he hears his mother calling, "Willie, bring me a drink of water," and he thought often that his mother was at the foot of his bed. He required constant watching and some restraint. He believed that those about him had entered into a conspiracy to kill his mother, and to get the insurance money. He talked incessantly at night, although fairly quiet during the day, but was utterly confused.

Examination by Dr. Hawke on November 25, 1908, was as follows: The patient usually repeated questions before attempting to answer, and his answers were always irrelevant. He showed general confusion, and answered only one or two questions intelligently or clearly. There was a vague realization where he was, but he was unable to give any definitely clear information about his life in the hospital. He was quiet and obeyed simple commands.

These cases require no special comment, but illustrate several familiar types of mental symptoms which have an etiological relation with syphilis. The important phase of the whole subject seems to me to be the early recognition of the psychic symptoms of syphilitic origin with a view of instituting the proper treatment early and before the process has progressed so far that therapeutic measures are hopeless.

I take pleasure in thanking Dr. Hawke for the privilege of studying some of these cases at the Philadelphia Hospital for the Insane.

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THE EFFECT OF STOVAIN ON THE NERVOUS SYSTEM

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As stovain produces temporary anesthesia, it occurred to us that repeated injections of the drug probably would produce anesthesia of gradually increasing duration until finally we might obtain a persisting loss of sensation, which would be the result of organic change. Whether the same may be said of any other drug has not as yet been determined. We desired to ascertain whether a systemic degeneration of the posterior roots and their continuation in the posterior columns of the cord is the common result of the repeated use of stovain. It was important, moreover, to determine whether the paralysis that occurs in stovain anesthesia is of a motor or sensory type, i. e., whether it is produced by changes in the peripheral motor neurons, or is the result of abolition of all afferent impulses which normally pass over the posterior roots. Reflex action and all recognition of tonicity or relaxation of the limbs, and necessarily of the position of the limbs, are lost if all peripheral afferent impulses are cut off.

Some investigations regarding the effects of stovain on the nervous system have been made, but they are not numerous, and in certain respects the results do not fully correspond with those obtained by us.

Spielmeyer found changes in the cells of the anterior horns from stovain poisoning in man, the dog and the ape, of the character of reaction at distance. In man the cells of the spinal ganglia were not affected. He regards the cellular changes as primary notwithstanding their character. The alteration was seen only in scattered cells. He has never seen degeneration of the white matter in the cord of man: at most he has found changes in the nerve cells of retrograde type. He has had five cases, three in apes and two in dogs, in which the degeneration was confined to posterior root fibers and their continuation in the cord, so that it would appear that stovain degeneration may at times be confined to the posterior roots. He seems to have depended on the Marchi method chiefly, and there is no mention of teasing of nerve fibers and of staining them in the fresh state. The degeneration affected the short fibers of the posterior roots, the reflex collaterals more than it did the long fibers. The stovain, he thinks, attacks the extramedullary portion of the posterior roots, and the degeneration of the intramedullary portion is secondary to this. This process differs from tabes, in Spielmeyer's opinion, because in tabes the degeneration begins in the intramedullary portion of the posterior roots.

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* From the Department of Neurology and the Laboratory of Neuropathology in the University of Pennsylvania.

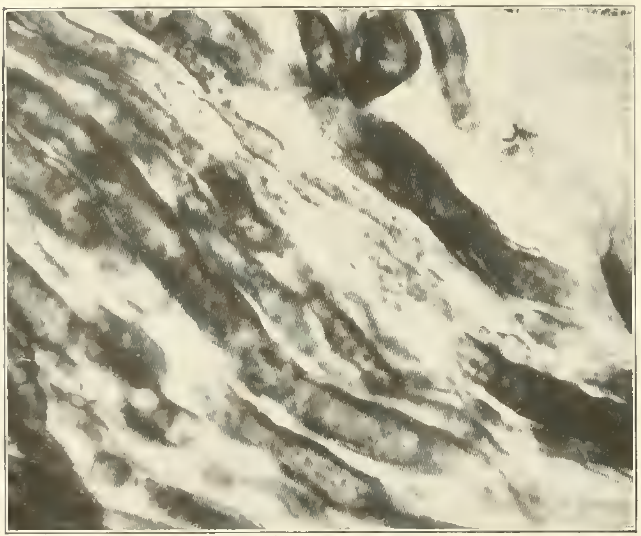

Fig. 1. Section through a lower spinal ganglion, showing degeneration as small black dots, in the nerve fibers.

Spielmeyer states that the posterior roots outside the dural sac were intact, in contrast to the greatly degenerated cauda equina within the dural canal. In the spinal ganglia not only the peripheral but also the central nerve fibers appeared normal by osmic acid stain, so that changes were not noticed in the posterior roots until the roots had entered the intradural space and come into direct contact with the stovain. The degeneration of the anterior roots, in the cases in which the process was more extensive and was not confined to the posterior roots, began where the anterior roots made their exit from the cord, and the intramedullary portions of the anterior roots were intact. In both the posterior and anterior roots the products of disintegration were soon absorbed. The anterior roots were degenerated secondarily also in the portion beyond the dural sac, so that the anterior root in contact with a spinal ganglion frequently appeared degenerated while the ganglion and its nerve fibers at each end were intact. In ten experimental cases that Spielmeyer examined the posterior roots were predominantly or exclusively affected; he therefore con-

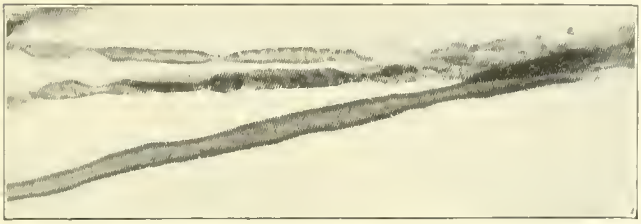

Fig. 2. Fibers of a lumbar posterior root teased and stained with osmic acid, showing degeneration.

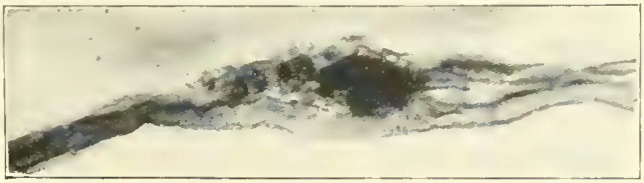

Fig. 3.—Fibers of a lumbar anterior root showing larger masses of degenerated myalin

cludes that stovain selects especially the posterior roots. He states that scar formation or inflammation plays no part; also that the degree of degeneration is not proportional to the amount of stovain used.

The changes in the cells of the anterior horns he found in man, as indicated in his illustrations, were pronounced. He found in animals swelling, displacement of the nucleus, and chromatolysis in the cells of the spinal ganglia. He thinks that the sensory and motor disturbances that occur after stovain anesthesia in man may be caused by similar changes in the spinal roots and in the periphery of the spinal cord, as are the temporary ocular muscle palsies from cellular changes; but pathologic studies are wanting in these cases.

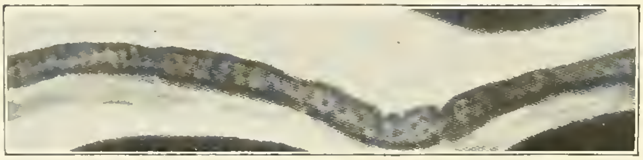

Fig. 4.—Fibers of a peripheral nerve from a hind limb, showing degeneration. In many fibers the degeneration was more advanced.

Root degeneration has not been found in man, although Spielmeyer[1] studied the specimens from sixteen cases. In half of these cases death occurred too soon to permit the Marchi method to be employed. These findings are remarkable, as root degeneration was obtained in all his injected animals. The symptoms following injections in apes were not very positive. In dogs he observed temporary paraplegia with disturbance of sensation and of micturition.

The investigation as conducted by van Lier[2] and Wossidlo,[3] showed that the cellular changes were temporary. Wossidlo found, after injection, paresis of the posterior limbs, restlessness, frequent respiration and clonic spasm of the posterior limbs. Usually these symptoms disappeared after artificial respiration. In some instances opisthotonos occurred. The lumbar region was examined one to twenty-four hours after the injection, and he found chromatolysis and swelling of the cell. When stovain was used the most pronounced changes were seen after one hour. Scarcely any normal cells were found in the anterior horns. The changes were more pronounced after two hours, and improvement was noticed in the cellular condition after six hours. The cells were normal after twenty-four hours. Van Lier's findings were similar. They used rabbits. Wossidlo concluded that tropacocain was less dangerous than stovain or novocain and that changes occurred in the cells in lumbar anesthesia, but that they were not serious enough to prevent the use of drugs in this method.

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[1] Spielmeyer: München. med Wchnschr., Aug. 4, 1908, p. 1629; Neurol. Centralbl., Jan. 16, 1909, p. 69.

[2] Van Lier: Beiträge zur klin. Chir., 1907, liii, 413.

[3] Wossidlo: Arch. f. klin. Chir., 1908, lxxxvi, 1017.

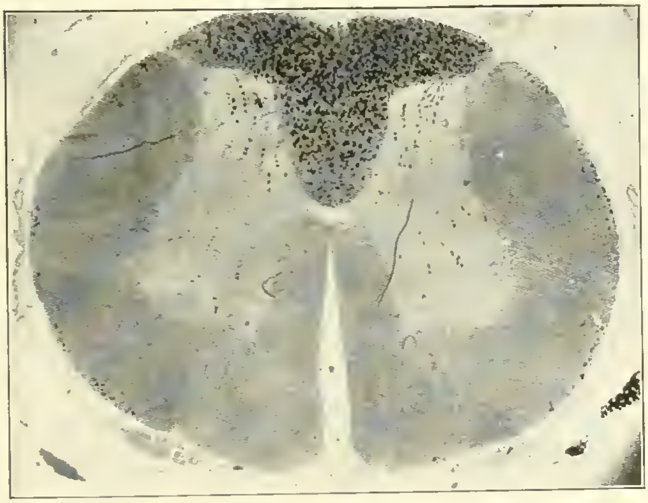

Fig. 5.—Section through the lumbar region (from retouched photograph) showing intense degeneration of the posterior columns.

Rehn could not demonstrate any changes in the cells of the anterior horns until after three days; then they were found in the lumbar, thoracic and cervical regions, and yet only a few cells in any one section were altered. The alteration began at the periphery of the cell, and the nucleus became peripheral. Animals that had lived ten to twenty-one days showed by the Marchi method, degeneration in the entire periphery of the cord and in the entire posterior columns, in the latter apparently systemic in character. Only cells in the anterior horns were affected; sensory cells were intact. Rehn's report[4] is of findings obtained by Klose and Vogt.

The technic of our experiments consisted in performing lumbar puncture on five dogs with careful avoidance of infection. The stovain solution used on Dogs D and E was boiled. The dose of each stovain injection varied from 0.05 gm. to 2 gm. and except in Dogs A and B. in each of which only one injection was given, the injections were usually at intervals of two to three days.

The symptoms may be divided into the temporary and the permanent. The temporary consisted of flaccid paralysis and complete or partial sensory loss. The hind limbs showed flaccid paralysis, while the entire body frequently showed the loss of sensation, implicating even the ear. Bladder and rectal control was lost, and the tendon reflexes were either diminished or absent. These symptoms occurred immediately or several minutes after the injection was given, and persisted from one to several hours.

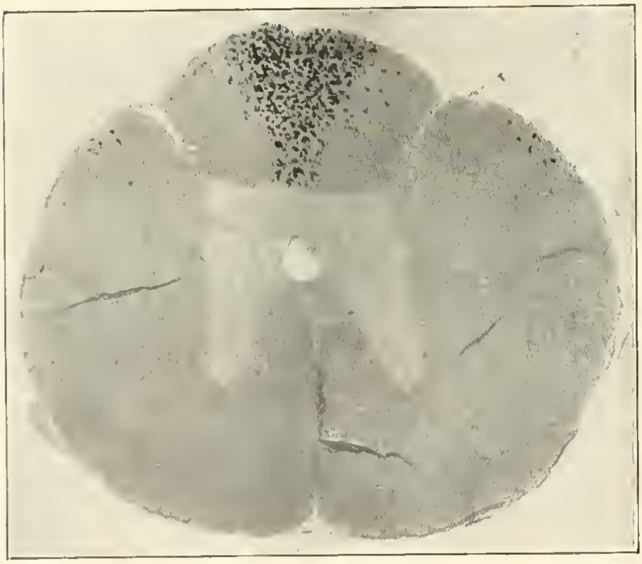

Fig. 6.—Section through the thoracic region (from retouched photograph) showing degeneration confined to the columns of Goll.

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[4] Rehn: Arch. f. klin. Chir., 1909, xc, 329

The permanent symptoms consisted of ataxia, decreased sensation, and, in Dog E, loss of patellar reflex. The symptoms became permanent after the third injection, remaining until the end of the experiment. The ataxia was recognized by the irregular gait, and the tendency to stand with the hind legs well apart, and the peripheral part of the hind limbs well on the ground. Sensory disturbances were demonstrated by the clip test. Dog A lived one hour after the injection, Dog B four hours, Dog C seventeen days, Dog D 2 months and Dog E forty-two days. The investigations have not been completed, but the results obtained are interesting.

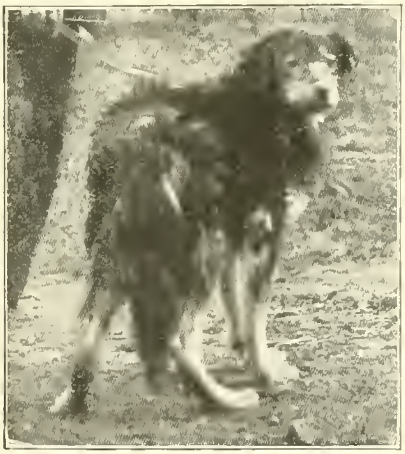

Fig. 7.—One of the dogs (Dog D) experimented on, showing ataxia of hind legs.

The alterations of the cells of the anterior horns were not important in any of the five dogs; in some slight central chromatolysis, and displacement and swelling of the nucleus were found. Dog D showed degeneration in the columns of Goll, more marked on one side, but dog E gave the most important results, and the following description is from the sections obtained in this case.

LUMBAR REGION

Axis cylinders in the periphery of the anterolateral and posterior columns were found here and there considerably swollen; some were of large size; swollen axis cylinders were seen in the root entrance zones of the posterior columns, but otherwise not in the posterior columns except near the periphery. A moderate degree of cellular reaction to the stovain was detected in the pia and roots of the cord in the form of round cell infiltration, but never as cells of polynuclear type. This is not a surprising finding and resembles what is seen in tabes. One would expect some cellular reaction to a poison affecting the nervous system as does stovain. A very slight perivascular round-cell infiltration was seen here and there in the cord; it was so slight as to be of no importance, and its existence was disputable. The swelling of the axis cylinders in the anterior and posterior roots was very distinct, and the swelling affected most of these axis cylinders. One forms the impression that the axis cylinders were more swollen in the anterior than in the posterior roots.

The posterior columns in the lumbar region were degenerated, as shown by the Marchi method, throughout a transverse section, but much less so in the ventral zones. The reflex collaterals were also much degenerated, and the degenerated fibers could be traced forward into the anterior horns. Small black dots were found along some of the anterior roots within the spinal cord, and this finding indicates a moderate amount of degeneration here. A slight degeneration was found by the Marchi method along the periphery in the anterolateral columns; it was far less intense than in the posterior columns. In the thoracic region the degeneration of the posterior columns was confined to the columns of Goll; the columns of Burdach seemed to be intact. The degeneration of the anterolateral columns in the thoracic region was insignificant. The lumbar sections were taken fully one and a half to two inches above the point of injection.

Anterior and posterior roots taken between the dural cavity and the posterior ganglia were teased in the fresh state and stained with a 1 per cent. aqueous solution of osmic acid. They presented considerable degeneration, chiefly in the form of minute black granules within the neurilemma sheaths, and the degeneration was more advanced in the anterior roots. One could not conclude from this finding that stovain affects the anterior roots more than the posterior; rather the finding would seem to imply that, the roots having been affected by the stovain within the dural sac, secondary degeneration would be found in the portion of the anterior roots examined, and retrograde degeneration in the portion of the posterior roots examined. Nerve roots taken from within the interior of the dural canal unfortunately were not examined by this method.

The nerve fibers in one of the lower spinal ganglia, placed in the fresh state in 1 per cent. osmic acid solution, showed intense degeneration of the fine granular variety. The cells of the ganglion presented little degeneration.

A nerve taken from the hind leg appeared intensely degenerated, when placed in the fresh state in osmic acid.

The method of studying nerve fibers in the fresh state with osmic acid, employed by us, does not appear to have been used in previous studies with stovain injections; it has given most satisfactory results. It is probably because of the employment of this method that we have obtained distinct degeneration in the posterior roots between the dural cavity and the ganglion as well as in the ganglion itself—findings not obtained by Spielmeyer. Our results differ from those obtained by others in the absence of important alteration of the cells of the anterior horns, although we employed the thionin stain for examining these cells.

It seems clearly demonstrated that stovain affects especially the anterior and posterior roots; the degeneration of posterior root fibers in our sections was intense; what is worthy of note, the degeneration of the intramedullary portion of the lumbar and sacral posterior root fibers in the thoracic region was still intense. The posterior thoracic roots were unaffected. Stovain evidently also causes slight degeneration in the periphery of the anterolateral columns, but has less effect here than on the nerve roots.

These lesions obtained by us could not have been produced by the trauma of the needle, as the sections of the lumbar region examined were one and a half to two inches above the point of injection, and yet the posterior and anterior roots were greatly degenerated.

It would be unwarranted to apply these findings too strictly to man, as no grave changes have been found as yet in the human spinal cord. At most our findings would show that repeated injections of stovain might be injurious, and would make one cautious in employing several injections within a short time in the same subject. We do not know whether stovain has more effect on the nervous system of the dog than on that of man.

The investigations show further that the paralysis produced by stovain is of the motor type, as the anterior roots were greatly degenerated.

Note on Correcting the Proof.—Investigations made with magnesium sulphate have not given clinical results similar to those obtained with stovain. Four injections of ten drops each of a saturated solution of this drug have been made into the dural cavity of a dog, extending over the period from March 31 to the present time. No ataxia has occurred, the dog walks with the hind legs close together, and the reflexes are normal, but sensation of the hind legs seems to be slightly diminished.

We are indebted to Dr. Allen J. Smith for the photographs.

1906 Chestnut Street—1632 North Franklin Street.

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FRIEDREICH'S ATAXIA[1]

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Friedreich's ataxia has been considered usually as a disorder confined to the spinal cord and cerebellum, but more thorough study reveals alteration of the cerebrum, peripheral nerves and muscles. The heart frequently has been found diseased, and was so in the case reported in this paper. It seems probable that careful examination would show more extensive implication of the body than is supposed to exist in the family diseases of the nervous system. The relation of the various family organic nervous diseases to one another is a question also worthy of consideration; and muscular dystrophy may be closely related to them. In some instances when the cerebellum in Friedreich's ataxia appears to the naked eye to be normal, microscopical study reveals great alteration of its nuclei. The cells of the anterior horns of the spinal cord may present degenerative changes. Fatty degeneration detectable within the muscle fibers by the Marchi stain; degeneration of the sensory roots of the trigeminal nerves; the presence of numerous naked axis cylinders shown by the Bielschowsky stain in the greatly degenerated columns of Goll, are findings obtained in the case reported in this paper, and so far as I know, not previously observed. There is need of explanation for the integrity of sensation when the posterior columns are so greatly altered.

The following notes were taken while the patient was in the service of Dr. Mills and from my observations after the death of the patient. She was in my service five days after I came on duty at the hospital.

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[1] Read at the thirty-sixth annual meeting of the American Neurological Association, May 2, 3 and 4, 1910.

H. McC—, laboratory number 514, female, white, age 24 yrs.; was admitted to the Philadelphia General Hospital, December 19, 1909, and died January 5, 1910. Her father died at the age of 57 from uremia. Her mother died from insanity. Two brothers are living; one sister died in childhood, and was physically defective. One of the patient's near relatives had the same peculiar gait that the patient had. A paternal uncle was paralyzed.

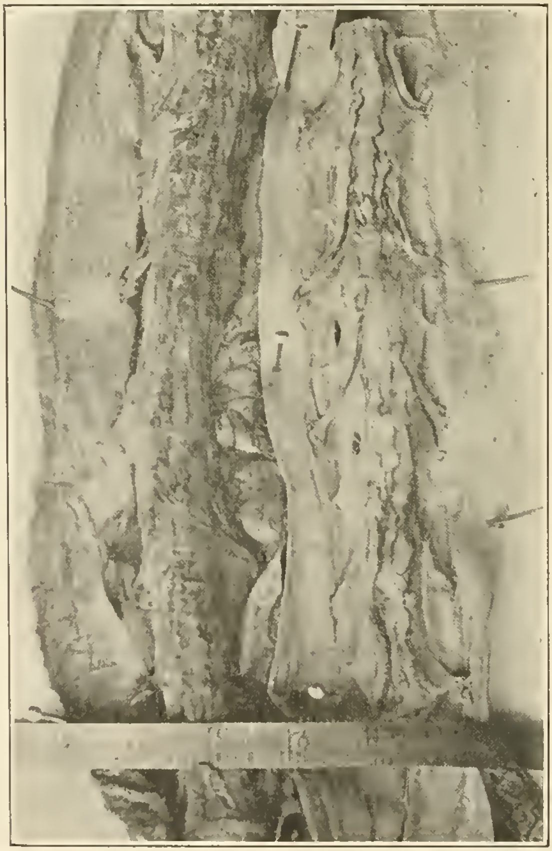

Fig. 1. Spinal cord from a case of Friedreich's ataxia (on the left), (Case I.). Compare this with the normal cord on the right.

The symptoms were first noticed when the patient was 7 years old. She staggered then as though drunk and the staggering increased gradually. At the age of 10 she was able to stagger around and run a little; she could still walk when 12 years of age, and when 15 years old was able to walk by supporting herself by leaning against objects. She had not been able to walk since she was 18 years of age. She has never had pain, except recently while in the hospital, when she complained of pain below the knees. The pupils are equal and regular; the irides react to light and in convergence. The eyeballs show incoördination in following an object. Intelligence is not very good. Her speech is defective and slow, and articulation is poor. The muscles of the upper limbs appear unusually well developed, and yet these limbs are very weak. The condition suggests pseudomuscular hypertrophy. When she is told to put a finger of either hand to the nose she raises the upper limb to about a right angle with the trunk, and the hand then falls to the nose with much ataxia, as though she had not the power to control its movements. The grasp of each hand is weak. The upper arms are proportionally better developed than the forearms. The heart sounds seem to be normal. Pain, temperature and tactile sensations are normal everywhere so far as they can be determined from the impaired mentality.

The lower limbs are entirely paralyzed and flaccid, and the patient can not move them in bed. The thighs are well developed, but the legs below the knees are distinctly wasted. The sphincters of the bladder and rectum are not implicated. The patellar reflexes are absent. Babinski's reflex is positive on each side. The peculiar deformity of the big toe, characteristic of Friedreich's ataxia, is distinct on the right side, less distinct on the left side. The Achilles reflexes and the biceps and triceps tendon reflexes are absent. The feet are in the position of talipes equino-varus.

The necropsy was performed by Dr. H. S. Wieder. I take this opportunity of thanking him for the care with which it was performed, as it is owing to him that pieces of many peripheral nerves and muscles were obtained.

Pathological Diagnosis.—Dilatation and cloudy swelling of the heart, moderate edema of the lungs, anemic infarct of the kidneys, acute nephritis, congestion of the pancreas.

The heart is about half again the normal size. The right auricle is distended with a blood clot. The heart muscle is normal in thickness but pale. The tricuspid and pulmonary valves are normal. The left ventricle is markedly dilated but the muscle is normal in thickness. The mitral and aortic valves are normal. The aorta and coronary arteries are normal.

The posterior roots to the naked eye are very much smaller than the anterior roots everywhere in the cord.

The brain, after it has been in 10 per cent. formalin a few weeks, and small pieces have been taken from each paracentral lobule, from one lateral cerebellar lobe, and from the medulla oblongata, for microscopical examination, weighs 1224 grammes.

The spinal cord and medulla oblongata are exceedingly small, but the cerebellum is of good size.

The upper part of the ascending frontal and the ascending parietal convolutions are narrow, especially on the right side of the brain.

Sacral and Lumbar Regions.—The posterior roots are much degenerated and when they are stained by the Weigert hematoxylin method very few nerve fibers are present. Clarke's columns are absent. The posterior columns are much degenerated, except in the anterior portion next to the gray matter, and the degeneration extends to each posterior horn. The posterior horns do not contain nearly so many fibers as normally should be present, and those found within them come chiefly from Lissauer's zones. The crossed pyramidal tracts are slightly degenerated. The anterior columns are normal. The cells of the anterior horns are about normal in number, and most of them are well formed, but in a portion of the lumbar region some much altered cells are found; in these the nucleus is swollen and the chromophilic elements and dendritic processes have disappeared. A fairly large number of axis cylinders are found throughout the posterior columns, even in the most peripheral part, by the Bielschowsky method. The posterior lumbar roots by this method show many fine axis cylinders.

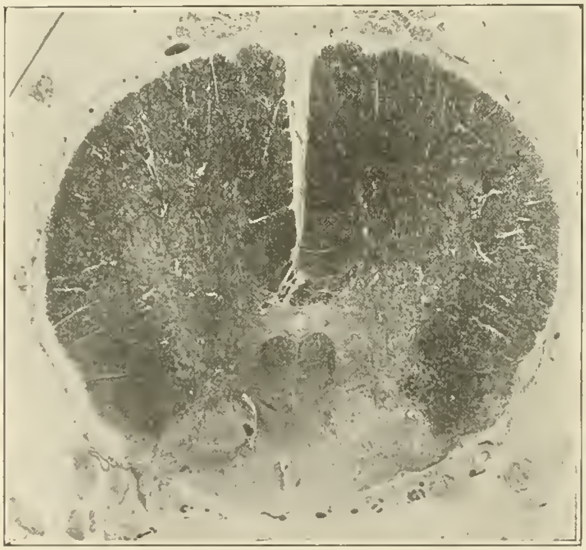

FIG. 2. Section from the lumbar region in a case of Friedreich's ataxia (Case I). The posterior columns are much degenerated.

Midthoracic Region.—The right direct pyramidal tract is degenerated. The crossed pyramidal tracts and direct cerebellar tracts are much degenerated. Gowers's tracts are not affected. The posterior columns are much degenerated, and the degeneration leaves only a few normal fibers close to the posterior horns. Lissauer's zones are intact. The posterior roots are much degenerated, but not as much so as are those in the lumbar region, and in neither region can the fibers preserved be considered as very fine. Sections obtained by Mallory's neurogliar method show dense sclerosis in the anterior half of the posterior columns, and the "tourbillons" described by Dejerine and Letulle are numerous. The central canal is a slit elongated antero-posteriorly. In each posterior horn where the column of Clarke should be is an area of dense neuroglia corresponding in size to this column, and fairly well defined. Longitudinal sections through the columns of Goll, from which the remainder of the posterior columns had been removed, in the lowest thoracic region stained by the Bielschowsky method, show many axis cylinders. In the posterior part of the sections the axis cylinders are longitudinally arranged, but in the more anterior part of Goll's columns they run obliquely, transversely and longitudinally, and form more or less of a feltwork, because of the "tourbillons." There are few axis cylinders in the extreme posterior part of the sections, but many in the middle and more anterior part of Goll's columns.

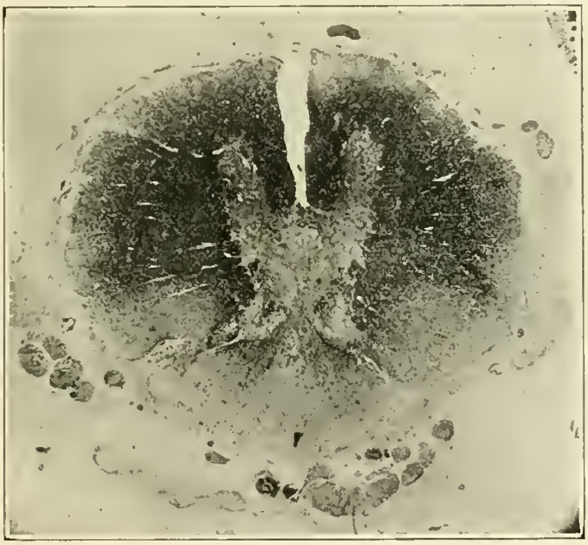

FIG. 3. Section from the mid-thoracic region in a case of Friedreich's ataxia. (Case I.) The posterior columns, the crossed pyramidal tracts, the direct cerebellar tracts, and the right direct pyramidal tract are much degenerated.

Cervical Swelling.—The posterior roots are much degenerated, but not nearly so much as in the lumbar region. The anterior roots are normal. The columns of Goll are much degenerated but contain a few scattered nerve fibers, too few for any function as shown by the Weigert hematoxylin stain. The columns of Burdach are moderately degenerated. The direct cerebellar and crossed pyramidal tracts on each side are moderately degenerated. Gowers's tracts are intact. Degeneration of the right direct pyramidal tract is distinct, the left tract is intact. The cells of the anterior horns do not appear so numerous as in most spinal cords, but are well formed. The pia and blood vessels of the pia are not affected anywhere in the cord.

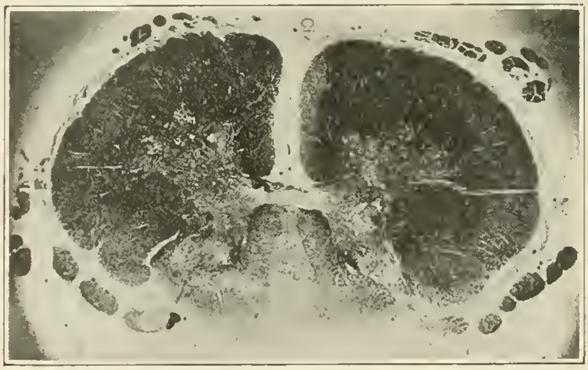

FIG. 4. Section from the cervical region in a case of Friedreich's ataxia. (Case I.) The columns of Goll are intensely degenerated, the columns of Burdach are less degenerated. The dirct cerebellar tracts, the crossed pyramidal and right direct pyramidal tracts are considerably degenerated.

Mallory's stain shows numerous "tourbillons" in the posterior columns. The sclerosis is much more intense in the ventral two thirds of Goll's columns. Some slight indication of the "tourbillons" is found in the crossed pyramidal tracts. The sclerosis is much more dense in the columns of Goll than elsewhere. The vessels of the posterior roots in the cervical and lumbar regions are much congested. The posterior columns stained by the Bielschowsky method show a fairly large number of axis cylinders; these are short and are cut longitudinally in tranverse sections because of the distortion of the fibers in the "tourbillons." These axis-cylinders are more numerous in more anterior portions of Goll's columns than they are in the extreme posterior part.

The right and left paracentral lobules stain well by the Weigert method. The nerve cells do not appear so large as normal, and possibly are not quite so numerous. No large Betz cells are found, although a few cells of the Betz type but smaller than these are present.

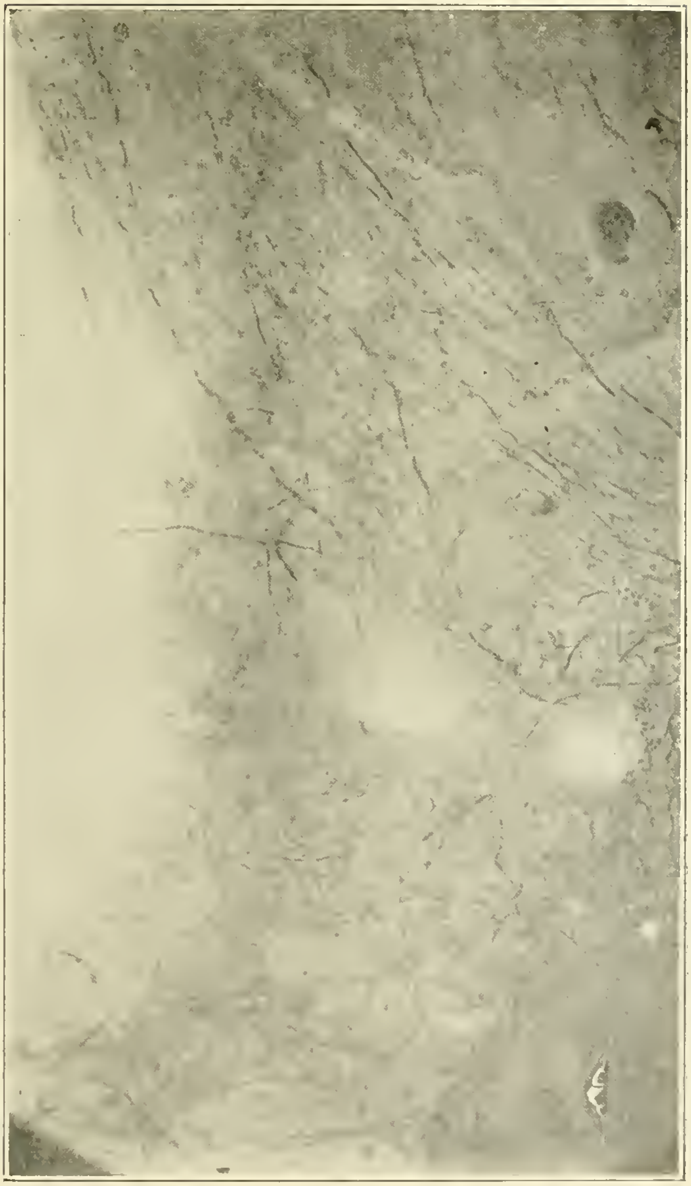

FIG. 5. Longitudinal section from a case of Friedreich's ataxia (Case I), taken from the low thoracic region and stained by the Bielschowsky method. Whereas in the posterior part of the columns the axis cylinders are seen arranged longitudinally, here in the anterior part of the columns of Goll the axis cylinders run in all directions. Compare with Fig. 6.

The cells of Purkinje in the cerebellum by thionin are numerous and well stained, and in sections made by the Weigert medullary method they appear normal. The nucleus dentatus contains comparatively few cells, and most of those present are shriveled. The superior cerebellar peduncles are smaller than usual.

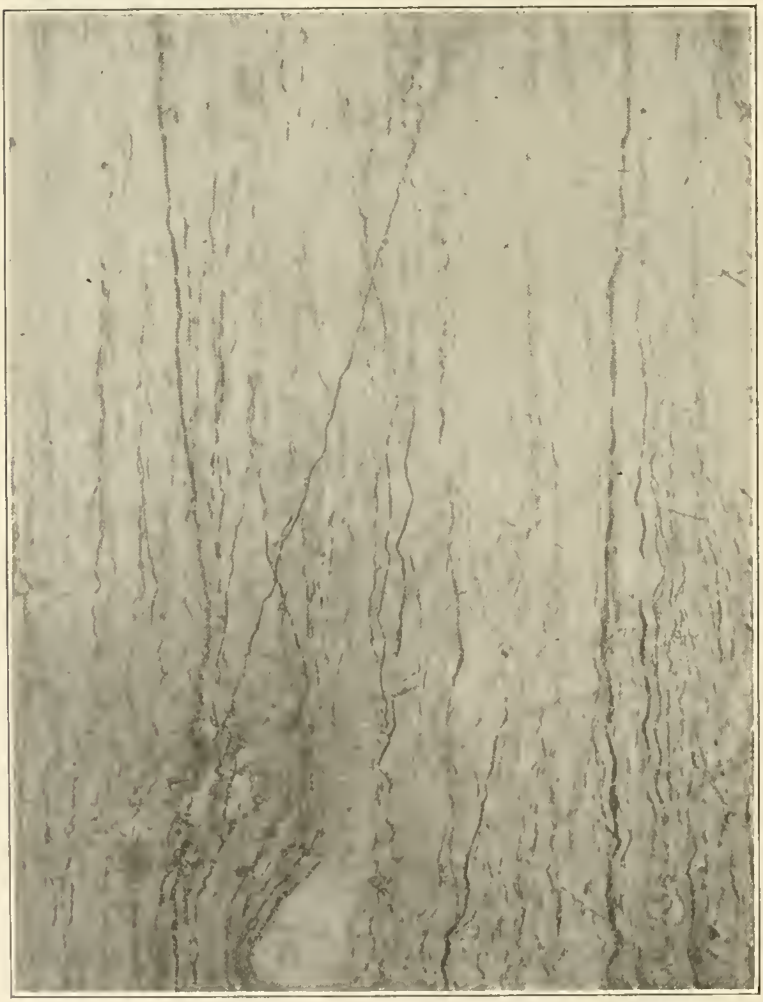

FIG. 6. Longitudinal section from a case of Friedreich's ataxia (Case I), taken from the low thoracic region and stained by the Bielschowsky method. Many naked axis cylinders arranged longitudinally are seen, whereas by the Weigert method this region appeared almost entirely degenerated. The axis cylinders are within the columns of Goll.

Sections were examined from the left radial, left ulnar, left median, posterior interosseous, external and internal plantar, and anterior tibial nerves; they are greatly degenerated, and the fibers preserved are not unusually small.

Sections were taken from the following muscles: extensor communis digitorum, tibialis anticus, extensor proprius hallucis, from the plantar region, including the interossei, abductor minimi digiti, thenar and hypothenar eminences, extensor primi internodii pollicis, and extensor secundi internodii pollicis.

The muscles show considerable alteration. Many of the muscle fibers are atrophied, the sarcolemma nuclei are increased in number, the transverse striation in some is indistinct, as is also the longitudinal striation to some extent, and some fibers have a hyaloid appearance. The nerve fibers found within the muscles show degeneration. There is no vacuolation of the muscle fibers. The extensor communis digitorum does not appear so much altered as the other muscles. The fibers of the extensor proprius hallucis are larger than those of other muscles. Muscle from the hypothenar and thenar eminences stained by the Marchi method shows many fibers with small black dots in lines within the muscle fibers, indicating recent degeneration. This is present only in moderate intensity in the tibialis anticus muscle.

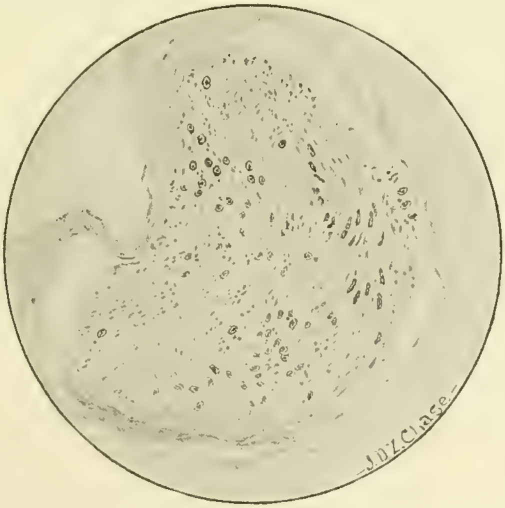

Fig. 7. Section from the anterior tibial nerve showing great degeneration.

Lumbar and Sacral Ganglia.—The cells of the capsules are much proliferated, many of the nerve cells are much shrunken, and many are highly pigmented. With thionin many of the nerve cells are pale and have peripheral nuclei. Medullated fibers are not very numerous within the ganglia, but are equally numerous at the two ends of a ganglion. A radicular nerve from a lower ganglion shows slight cellular infiltration.

Gasserian Ganglion.—Some proliferation of the cells of the capsules is found but not so much as in the lower spinal ganglia. There is a distinct increase of round cells between the nerve cells. The nerve cells are not much altered except that some are much pigmented. The sensory roots of both fifth nerves are much degenerated. Some bundles are deeply stained by the Weigert method, and these probably belong to the motor portion. The spinal roots in the medulla oblongata, however, show little alteration.

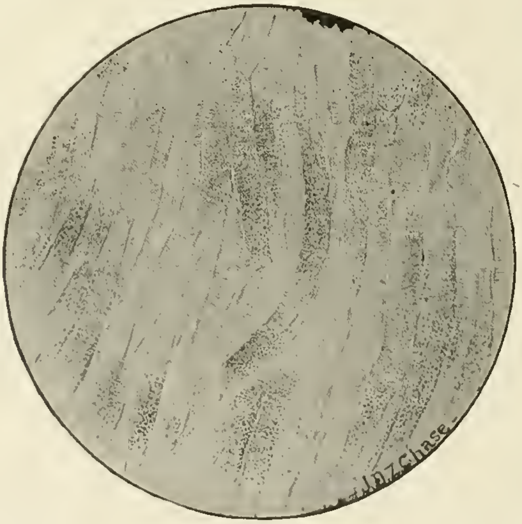

FIG. 8. Section from the extensor communis digitorum muscle, stained by the Marchi method. Numerous small black dots, caused by fat within the muscle fibers, are seen.

The medulla oblongata shows no degeneration of the anterior pyramids. The cells of the lower olive are normal in number but are much smaller. The optic nerves and chiasm are not degenerated.

I am indebted for the photographs illustrating this case to Dr. Allan J. Smith.

The following clinical case of Friedreich's ataxia has been in my service frequently at the Philadelphia General Hospital, and has been studied also by my colleagues on the neurological staff. It is included because of the pronounced atrophy of the limbs.

W. Soudan, 41 years old, began to walk when three years old, and walked with a cane until he was 15 years old. He staggered much and was clumsy in lifting his feet, and often fell when walking. In 1895 he became unable to walk, and for some time before his admission he found it more difficult to walk at night.

In 1890 he had severe attacks of pain in the stomach, with nausea and vomiting, suggesting gastric crises.

Speech always has been slow. Intelligence is good.

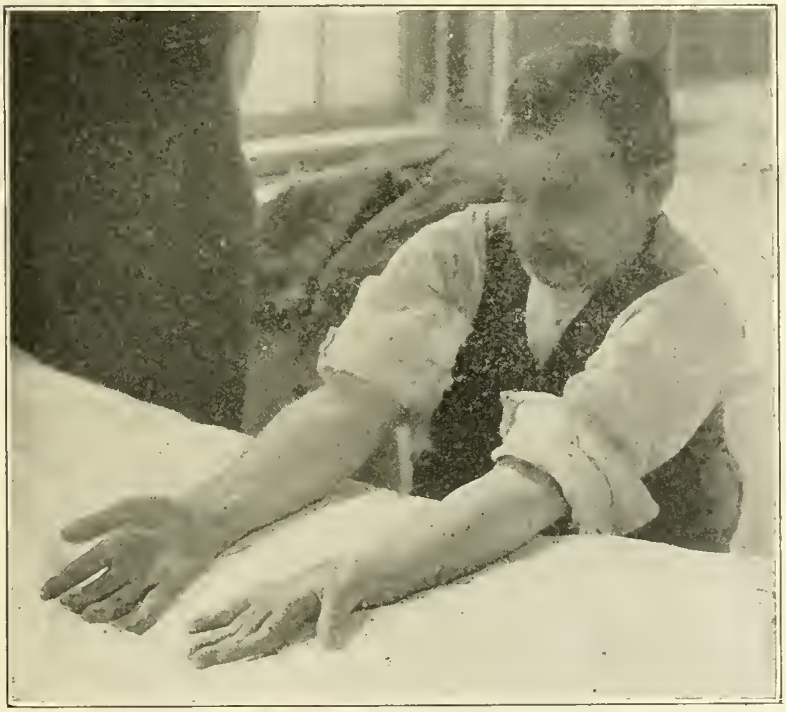

Fig. 9. Pronounced atrophy of the hands in a case of Friedreich's ataxia. (Case II.)

His condition since 1898 has varied very little, except that he has grown weaker in his lower limbs. In 1898 he had marked atrophy of the interossei muscles and of the thenar and hypothenar eminences, and marked symmetrical atrophy of the calves. His station was very ataxic.

In May, 1905, Babinski's sign was absent on both sides. Friedreich's toe was not present.

His condition at present (March 2, 1910) as observed by me is as follows: He has marked nystagmus in looking either to the right or left, but none in looking straight forward. There is no tremor of the head, and no paralysis of the muscles of the face: The tongue is normal. Touch, pain, heat and cold sensations are normal in the face. Speech is slow and somewhat monotonous. The pupils are equal, although the left may be a trifle larger. The irides respond promptly to light and in convergence.

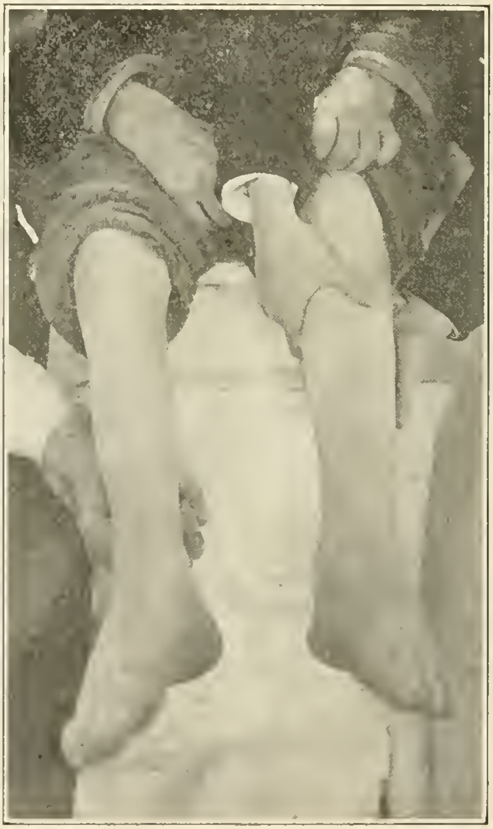

FIG. 10. Pronounced atrophy of the feet and legs below the knees, with foot-drop and hard edema of the feet in a case of Friedreich's ataxia. (Case II.)

Touch, pain, heat and cold sensations are normal in the upper limbs, even in the hands. The hands and lower part of the forearms are greatly wasted, and the fingers except the thumbs are partially flexed. From the middle of the forearms upward the upper limbs are well developed. Considerable fibrillary tremor is seen in the triceps muscles and in the right second interosseous space. The biceps and triceps tendon jerks are absent on each side. He has good motor power at the shoulder and elbow, and considerable power at the wrist. The grasp is surprisingly strong in view of the atrophy of the hands. Finger to nose test shows marked ataxia. He recognizes objects in his hands very imperfectly. Sense of position is good in each hand.

The power in the lower limbs is very good at hips and knees. The lower limbs below the knees are greatly wasted. The feet drop at the ankles, and the muscles of the feet are very flaccid. The feet are cyanotic and show a considerable degree of hard edema, the flesh not pitting on pressure. He has much ataxia in trying to touch an object with either foot.

Patellar and Achilles reflexes are lost on either side. Pain, heat and cold sensations are normal in the lower limbs, even in the feet. There is moderate impairment of tactile sensation and of the sense of position in the feet.

When supported by the arms he is able to stand and to bear a part of his weight on his lower limbs, but his steps are very incoördinate. He has not the Friedreich's toe.

The first case is especially worthy of comment in the atrophy of the legs below the knees (and atrophy in peripheral parts of all the limbs was very pronounced in the second case), in the pronounced degeneration of many nerves examined, and in the condition of the upper arms, and to a less extent of the thighs, resembling pseudohypertrophy, and associated with great weakness.

Atrophy of the limbs occurs in Friedreich's ataxia but is not common; its existence is denied by some writers, thus Otto Maas[2] in 1904 reported two clinical cases in brother and sister, in which he excluded Friedreich's ataxia because of spasms and pronounced atrophy which in his own words "diesem Symptomencomplex fremd sind."

Bäumlin,[3] however, in 1901 reported a case of Friedreich's ataxia without necropsy with atrophy of the interosseous muscles of the hands and pseudohypertrophy of the shoulders, upper arms, and forearms, with fibrillary twitching. He remarked that atrophy limited to muscle groups is rare in Friedreich's ataxia; he refers to cases reported by Dejerine, Hodge, Whyte, Bramwell, Griffith, and Rook, and he quotes a number of authors who have found degeneration of the peripheral nerves in Friedreich's ataxia, but states that necropsies have not been obtained in cases with atrophy.

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[2] Maas, Berliner klinische Wochenschrift, no. 31, Aug. 1, 1904, p. 832.

[3] Bäumlin, Deutsche Zeitschrift für Nervenheilkunde, vol. 20, 1901, p. 265.

Kollarits,[4] as late as 1906, made the statement that muscular atrophy does not belong to Friedreich's ataxia. He seems to have changed his opinion to some extent later, as in 1908 he wrote that the muscular atrophy of this disease is caused by changes in the cells of the anterior horns or peripheral nerves. Disease of the peripheral nerves has only occasionally been found, and this can not be always the cause of the muscular atrophy. There are cases, he says, like Schultze's, his own and many others in which muscular atrophy occurs in Friedreich's ataxia, although the cells of the anterior horns, the anterior roots and the peripheral nerves are healthy. As the muscular atrophy is not the result of disease of the peripheral neurones we must seek another cause. It occurs only in advanced cases, and it might be attributed to inactivity, but that is not a probable cause. He attributes the atrophy to muscular dystrophy; this opinion is strengthened by his case and others as the dystrophy was observed histologically.

One can now hardly dispute that atrophy occurs in Friedreich's ataxia, but it seems to be only a late sign. It is important to determine the cause of this atrophy. As mentioned above, Kollarits is unwilling to attribute it always to disease of the nerves, anterior roots, or cells of the anterior horns. When Dejerine[5] wrote in 1890 on the disease under consideration he could state that no definite information was obtainable on the condition of the peripheral nerves. Rütimeyer is the only author to whom he refers in this matter, and he found only a few fibers degenerated in the nerves. Dejerine believed that degeneration of nerves does not occur in Friedreich's ataxia as it does in tabes.

Oppenheim in the fifth edition of his text-book states that atrophy of the posterior roots and nerves is not a constant nor important finding.

Jules Vincent[6] in his thesis published in 1900 states that usually the cells of the anterior horns are found to be normal, but Friedreich, and Rütimeyer, in certain cases found them atrophied, and Vincent believed these changes may explain the muscular atrophy. Bäumlin[7] in 1901 stated that twenty cases of Friedreich's ataxia with necropsy were on record, and that in six of these changes in the anterior horns were found, and he gives reference to these.

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[4] Kollarits, Deutsche Zeitschrift für Nervenheilkunde, vol. 30, 1906, p. 295.

[5] Dejerine, Comptes rendus de la Société de Biologie, 1890, p. 105.

[6] Jules Vincent, thesis, Paris, 1900

Bing[8] states that in necropsies in cases of Friedreich's ataxia neuritic changes were found by Friedreich, Rütimeyer, Guizzetti, Mirto, Bonnus, Mackay; and that these changes were chiefly in the sciatic nerve. He attributed the atrophy below the knees in his case with necropsy to neuritis, although if the lesions had been studied earlier in the case he thinks they might have resembled the findings of muscular dystrophy.

Dejerine and Thomas[9] in 1907 found the nerves degenerated, and the smaller fibers in the nerves were in excess. The cells of the anterior horns were normal.

Mott's[10] case is one of the most thoroughly studied in regard to Friedreich's ataxia in the literature. He found the anterior roots and the cells of the anterior horns at all levels normal, but he found a great outfall of coarse fibers with integrity of fine fibers in the peripheral nerves, especially in the nerves of the lower limbs, and wasting of muscles although striation was fairly normal. Mott adds that only in a few cases of this disease have the peripheral nerves been examined, in some of these the nerves were reported to be normal, in others degenerated, "Recent chromolytic changes" in the cells of the anterior horns which Mott found he regards as unimportant.

Müller[11] in a case he reported found no decrease in the number of cells in the anterior horns, although he states that these cells were few in some of the cervical sections.

The question of atrophy is closely associated with that of muscular dystrophy occurring in Friedreich's ataxia. Bäumlin in 1901 reported the finding of pseudohypertrophy of the shoulders, upper arms and forearms, with fibrillary twitching. He regarded pseudohypertrophy as very rare in Friedreich's disease and even questioned whether it can be said to have occurred with certainty. The cases of Mastin, in which the pseudohypertrophy was chiefly in the calf muscles, Bäumlin[12] regarded as transitional forms.

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[7] Bäumlin, Deutsche Zeitschrift für Nervenheilkunde, vol. 20, 1901, p. 265.

[8] Bing, Deutsches Archiv für klin. Med., vol. 83, 1905, p. 199.

[9] Dejerine and Thomas, Revue Neurologique, vol. 15, 1907, p. 41.

[10] Mott, Archives of Neurology from the Pathological Laboratory of the London County Asylums, vol. 3, 1907, p. 180.

[11] Wladislaus Müller, Wiener klinische Rundschau, 1908.

Bing[13] writing in 1906 remarked that pseudohypertrophy of the shoulder girdle, of the upper arms and forearms, seen in his case, had not previously been observed in any case. He does not appear to have given proper credit to Bäumlin. Bing regarded the condition in his case as muscular dystrophy, and believes it was not the mere occurrence of muscular dystrophy with Friedreich's disease in the same person, but that a close connection existed between the two diseases. He depended largely on the histological examination for the diagnosis of muscular dystrophy. There is close connection ("an unbroken chain" he calls it) between Friedreich's disease, hereditary cerebellar ataxia, family cerebral diplegia, family spastic paralysis, family lateral sclerosis, etc.

In this statement he follows closely in the footsteps of Jendrássik.

Jendrássik[14] believes the hereditary diseases are related diseases and have only an apparently external similarity with certain exogenous disease types, as tabes, spastic paralysis, Huntington's chorea, etc. There is no close relationship between the exogenous and endogenous, *i. e.,* hereditary diseases. The number and variety of the types of hereditary diseases are great, and they pass one into the other, and make every classification impossible. Jendrássik tries to do with the hereditary diseases what Erb has done with the types of muscular dystrophy, to unite them all under one head. He reports in this paper two cases of atypical Friedreich's disease with weakness of the shoulder muscles like that of dystrophy.

In his second contribution[15] to hereditary diseases, he says that hereditary nervous diseases have an identical course within the same family, but vary greatly in different families, so that one may describe almost as many types as there are families affected. He has included all under the head of the family degeneration, and speaks of the various forms as the dystrophic type, spastic paraplegic type, Friedreich's type, etc.

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[12] Bräumlin, Deutsche Zeitschrift für Nervenheilkunde, vol. 20, 1901, p. 265.

[13] Bing, Deutsches Archiv für klinische Medicin, vol. 83, 1905, p. 199.

[14] Jendrássik, Deutsche Zeitschrift für Nervenheilkunde, vol. 22, 1902, p. 444.

[15] Jendrassik, Deutsches Archiv für klinsche Medicin, vol. 61, 1898, p. 187.

Kollarits refers to the occurrence of muscular dystrophy with hereditary spastic paralysis, quoting Seeligmüller, Hoffmann, O. Maas, Jendrássik, and himself, and asserts that all these observations show that Jendrássik is correct in believing that muscular dystrophy, hereditary spastic spinal paralysis, Friedreich's disease, and Marie's cerebellar hereditary ataxia, are not independent diseases, but are inseparable and pass into one another as forms of the family heredo-degeneration.

Raymond and Ross,[16] in reporting a clinical case of family disease intermediate to spastic paralysis and hereditary cerebellar ataxia, refer to the cases of Strümpell, Newmark, Brissaud, Bischoff, in which a combined sclerosis was found. They also assert that all gradations may exist between the different types of family disease of the nervous system. The coexistence of myopathy with Friedreich's disease (Ghilarducci) gives a further application to this view.

Pathology affords some support to these radical views and is deserving of more attention in this respect than has been paid to it; thus degeneration of the posterior columns in combination with degeneration of the pyramidal tracts has been found in family spastic paralysis (Newmark).[17] The former is slight in the lumbar region, but becomes more pronounced in the thoracic region, and occupies the columns of Goll, and becomes intense in the cervical region. The cells of the columns of Clarke are not numerous. These cells have been found affected in many cases of combined system disease not of family form. The degeneration of the posterior columns differs from that of Friedreich's ataxia chiefly in being slight in the lumbar region. It would seem to be the result of death of the more terminal portions of the fibers in the columns of Goll, and the difference between Friedreich's ataxia and family spastic paralysis, so far as pathology is concerned, seems to be one largely of degree. The cells of Clarke's columns are greatly affected in the former, slightly in the latter. In the former the degeneration of the posterior columns predominates over that of the lateral columns; in the latter the degeneration of the pyramidal tracts predominates over that of the posterior columns, at least in the lumbar region. In each family group the tendency to degeneration is greater in the same tracts, but in the family spastic paralysis the degeneration does not appear to extend into the lumbar region to any severity. Newmark's two cases with necropsy show however that in family spastic paralysis the degeneration of the columns of Goll above the lumbar region may equal or exceed that of the pyramidal tracts.

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[16] Raymond and Ross, L'Encéphale, March 10, 1909, p. 209.

[17] Newmark, Deutsche Zeitschrift für Nervenheilkunde, Vol. XXVII, 1904, p. 1, and Vol. XXXI, 1906, p. 224.

It is noteworthy that in both the cases of Friedreich's ataxia I report in this paper, more especially in the second, the atrophy was confined to the peripheral parts of the limbs and resembled closely that occurring in neural muscular atrophy. In my second case the atrophy had not seriously implicated the central portions of the limbs even after many years. This restriction to the peripheral parts of the limbs is one of the most diagnostic features of the neural muscular atrophy.

Gierlich[18] regards neural muscular atrophy as a special form, but he acknowledges that transitional cases to muscular dystrophy, tabes and neuritis are to be recognized. We may take Gierlich's case as a typical example of the neural atrophy, and compare it with Friedreich's ataxia. A boy, apparently normal at birth, showed paralysis of the dorsal flexors of the feet at the beginning of the second year, with shortening of the Achilles tendons and formation of talipes equino varus. The hand muscles began to waste at the fourth year of life. The patellar reflexes were lost. Sensation and coördination were normal.

Microscopically was found degeneration of the posterior columns which occupied almost the entire area except the dorso-ventral fields, but higher was almost confined to the columns of Goll, invading the columns of Burdach slightly. Degeneration of less intensity was found in the postero-lateral portion of the lateral columns, *i. e.*, in the crossed pyramidal tracts, the direct cerebellar tracts, and the tracts of Gowers. Loss of fibers and atrophy of cells were observed in Clarke's columns. Lissauer's zone was intact. Some of the cells of the anterior horns were degenerated. All these changes were almost identical to those observed by me in my case of Friedreich's ataxia, except that in the latter the cells of the columns of Clarke had entirely disappeared, and the posterior roots were greatly degenerated, whereas in Gierlich's case of neural atrophy the latter were intact. The alterations of the muscles and nerves resembled that observed in my case. Degeneration of posterior roots has however been found in neural muscular atrophy (Marinesco); in other cases the posterior roots were intact (Siemerling, Sainton, Dejerine and Armand-Delille).

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[18] Gierlich, Archiv für Psychiatrie, vol. 45, no. 2, 1909, p. 447.

Gierlich also has been interested in the resemblance of the pathological findings of neural muscular atrophy to those of Friedreich's ataxia, and yet the characteristic symptoms of the latter, viz.: ataxia, nystagmus, speech disturbance, etc., are absent in neural atrophy. He does not fully discuss the subject.

It seems probable to me that one must seek the cause of the characteristic symptoms of Friedreich's ataxia in some additional changes to those mentioned. The alteration of the posterior roots and of the cells of Clarke's columns in great intensity is not common in neural atrophy and may help to explain the symptoms of Friedreich's ataxia. The ataxia, nystagmus and speech disturbances of the latter may find their explanation in the alteration of the cells in the cerebellar nuclei, especially in those of the nucleus dentatus, and possibly of those in the lower olive; changes which probably have often been overlooked in Friedreich's ataxia.

As opposed to these views in which the attempt is made to do away with all sharp distinctions between the family forms of disease are the statements of Steinert and Verse.[19] These authors say that it is not so well known that lipomatosis and pseudohypertrophy occur in neuritis. The nature of the muscular atrophy in Friedreich's disease can not be considered settled, but transitional cases of Friedreich's disease to primary myopathic progressive dystrophy, which destroy the sharp lines dividing the two diseases, do not exist. Only in one case from the Budapest clinic does the atrophy seem to be similar to the type of myopathic progressive dystrophy (Jendrássik) and this case may be a combination of the two diseases. They regard the attempt to do away with sharp distinctions between the hereditary diseases as unwarranted. They regard Bing's statement as rash, viz., that the pseudohypertrophy of the shoulder muscles and upper limbs was muscular dystrophy as shown by the microscopical examination. They state that pseudhypertrophy was found by Bing where the nerves were not diseased, but they have no explanation to give for this.

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[19] Steinert and Versé, Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie, vol. 21, no. 1, p. 105.

Jules Vincelet stated in his thesis (1900) that most authors have found the deep sensation normal, and that degeneration of the posterior columns was present in all cases. He found only eighteen cases with necropsy, but he did not include Griffith's cases. Mott recently (1907) has stated that all cases have shown degeneration of the posterior columns. Dejerine in 1890 mentioned that the degeneration of the posterior columns in Friedreich's ataxia does not always correspond to the degeneration of the posterior roots, especially is this true when Lissauer's zone is intact. In tabes the degeneration of the posterior roots is always proportional to that of the posterior columns. Because of the better condition of the roots in Friedreich's ataxia, and of the good condition of the nerves, the sensation is intact, according to his view. Since 1890 we have had reason to doubt the integrity of the peripheral nerves in advanced Friedreich's ataxia, and must seek another explanation for the preserved sensation. Dejerine in this paper made the assertion that intense degeneration of the posterior columns may exist a long time without sensory changes.

In the case reported by Dejerine and Thomas[20] in 1907 the ataxia was extreme, the sense of position was intact. They found the fibers in the posterior roots of fine caliber, but they speak of the finding as marked atrophy of the fibers, instead of stating, as Mott does, that the large fibers had degenerated and the small fibers were preserved in the posterior roots. In the posterior columns, except in the cornu-commissural zones, and the postero-external root zones, the axis cylinders had almost completely disappeared. Lissauer's zone contained many axis cylinders. In the cervical region the columns of Goll contained few axis cylinders by Cajal's method. They say their case shows that Goll's columns are not necessary for sensation. They found not only the central segment of the posterior roots, but also the portion peripheral to the ganglion atrophied. They believe the atrophy of the posterior roots (they never speak of small fibers being simply small fibers which have resisted degeneration, as Mott considers them) is a retrograde atrophy from the degeneration of the posterior columns.

In this case the sense of position was intact and Goll's columns were completely degenerated. Where then do fibers of sense of position and touch ascend?

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[20] Dejerine and Thomas, Revue Neurologique, vol. 15, 1907, p. 41.

In Kollarits's[21] case touch, temperature and pain sensations were intact notwithstanding the posterior columns were intensely degenerated. This is not a new observation but one that has not received sufficient attention. Friedreich said it is certain that the posterior columns are not the only or most important tracts for conduction of sensation. This subject has been studied also by E. Müller. Müller believed that naked axis cylinders in the degenerated areas conveyed the sensations, but he had no findings justifying this opinion. Kollarits says it is certain that the view concerning conduction of tactile sensation in the posterior columns is contradicted by many observations.

Mott believes the small fibers of the posterior roots subserve cutaneous sensory and viscero-vascular functions, and in Friedreich's ataxia there is little or no affection of cutaneous sensibility and no visceral disturbances. The fine fibers are lost in tabes, therefore the alteration of sensation in tabes occurs.

My second case shows that cutaneous sensation is not always absolutely intact, as tactile sensation in the peripheral portions of the lower limbs was affected, but sensory changes usually are slight in Friedreich's ataxia. This may be explained by the discovery in my first case of many axis cylinders by Bielschowsky's method in the posterior columns. The only other case of this disease in which a selective axis cylinder stain was employed was that reported by Dejerine and Thomas. In the cervical region in their case few axis cylinders were found in the columns of Goll by Cajal's method. In my case they were numerous by the Bielschowsky method in the middle and more anterior portions of the columns of Goll in the lower thoracic and cervical regions, and in the "tourbillons" they took part in these peculiar formations and were cut more or less longitudinally for a short distance in transverse sections. It is probable that the posterior columns are capable of more function in Friedreich's ataxia than the Weigert's medullary stain gives reason to believe.

Ataxia is a common sign of Friedreich's ataxia, and gives a part of the name. The presence of so much incoördination with preserved sensation is remarkable, but we have every reason to believe that fibers necessary for coördination are distinct from those on which sensation depends. J. S. Risien Russell,[22] in his recent Lettsomian lectures, describes the anatomy of the tracts of coördination in the following words:

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[21] Kollarits, Deutsche Zeitschrift für Nervenheilkunde, vol. 34, 1908, p. 410.

"In the dorsal sensory nerve roots are fibers which convey afferent impressions from the muscles, joints and deeper structures, as opposed to the skin, some of which pass to Clarke's column of cells, from which is derived the dorsal or direct cerebellar tract; while others cross in the anterior commissure, and pass upwards in the ventro-lateral region of the cord, constituting the ventral or crossed cerebellar tract of Gowers. Both of these tracts pass to the cerebellum to terminate in the cortex of the organ, the former, as we have already seen, by way of the inferior peduncle, while the latter courses along the superior peduncle to end in the cortex of the middle lobe. From the cortex fibers pass to the nuclei, and from the dentate nucleus fibers are derived which leave the organ by way of the superior peduncles, to reach the red nucleus and optic thalamus of the opposite side, and through the thalamus connect the cerebellum with the cerebral cortex."

While therefore there seem to be no fibers that pass directly from the direct cerebellar and Gowers's tracts to the cerebellar nuclei, these nuclei receive fibers from the cerebellar cortex in which these tracts end, and therefore may be atrophied secondarily to degeneration of these tracts.

In the recent experimental work of MacNalty and Horsley[23] lesions of the cervical spino-cerebellar tracts caused ataxia and clumsiness of movements, most noticeable in the upper limb. These fibers arise at least in part in Clarke's column.

The ataxia of tabes probably is largely the result of degeneration of fibers going to Clarke's columns, even though the cells of these columns do not disappear. The same conditions exist in Friedreich's ataxia with absence of the cells of Clarke's columns, as these cells seem to have disappeared in almost every case. Vincelet says Clarke alone found these cells normal.

Mott found degenerative atrophy of the dentate nucleus as I have also. He states that there was not a healthy normal cell in this nucleus; some of these cells were represented by a little pigmented protoplasm. The Purkinje cells were atrophied in places. There is thus a pronounced disturbance in the tracts of coördination in the cord (the direct cerebellar and tracts of Gowers), as well as of the chief organ of coördination, the cerebellum. The cerebellum, according to Oppenheim,[24] has been found normal in many recent cases; in others (Raymond, Mott), hypogenesis (unusual smallness) or abnormality of its structure, especially of its nerve cells, has been found. Müller, in a very comprehensive article on this disease, states that in by far the majority of cases the cerebellum was entirely normal, but one can not but question the thoroughness of the examination of some of these cases. The number of cases, he states in 1908, in which hypoplasia of the cord and cerebellum, as well as of their connections, with or without structural changes occur, is very small. If we collect the cases in which there were symptoms of disease of the cord and cerebellum dependent on congenital hypoplasia with or without degeneration, they number something over ten.

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[22] J. S. Risien Russell, British Medical Journal, Feb. 19, 1910, p. 425.
[23] MacNalty and Horsley, Brain, vol. 32, 1909, p. 237.

Almost all authors, according to Müller, have either neglected the examination of the cerebrum or have made no mention of its condition. Nonne found diminution of certain cerebral gyri in the brain of a man, but no microscopical changes; the brain he studied weighed 1,030 grammes. In another case, the brain of a man weighed 1,020 grammes, some gyri were small, but no microscopical changes were found in the brain. Cramer found slight widening of the fissures and slight atrophy of the gyri of the frontal lobe. Straussler found moderate diminution of the gyri, especially in the frontal lobe, and widening of the fissures and slight atrophy of the cerebral cortex with peculiar changes in the cells, unimportant in the cerebrum but important in the cord, cerebellum and medulla oblongata. These are not the only cases in literature, but Müller says these were collected from nearly 200 cases in the literature, and they show how little importance has been placed on the examination of the brain in Friedreich's ataxia. He refers to one or two other cases in which the cerebral lesions seem to be of little importance.

Müller[25] found many changes in the cerebral cortical nerve cells, the layers were not clearly marked, many cells were imperfectly formed, and the Betz cells were absent in the precentral gyrus (he does not mention whether they were found in the paracentral lobule). Müller found changes in the entire central nervous system.

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[24] Oppenheim, Lehrbuch, p. 224.

[25] Waladislaus Müller, Weiner klinische Rundschau, 1908.

Mott describes naked eye wastings of the upper part of the cerebral convolutions, atrophy of Betz cells, thinning of the ascending frontal and ascending parietal cortex, diffuse sclerosis of the internal capsule and of the pyramidal tract in the crus cerebri, and marked sclerosis of the pyramidal systems, crossed and direct, in the medulla oblongata and spinal cord. Mott's patient was feeble-minded and suicidal. He found some medium-sized Betz cells but almost complete absence of giant Betz cells.

My patient was also rather feeble mentally, but many cases of Friedreich's ataxia present no mental deterioration. The upper part of the ascending frontal and ascending parietal convolutions were narrow and no large Betz cells were found in the paracentral lobules.

From findings like these it is difficult to accept the theory that the degeneration of the lateral columns is not in the crossed pyramidal tracts. Oppenheim[26] regards the changes described by Mott in the cells of the motor cortex as probably secondary, but there seems to be no good reason for so regarding them.

The degeneration of the direct pyramidal tract is another argument in favor of the implication of the pyramidal tract fibers. Oppenheim says Schultze once found the direct pyramidal tract degenerated. In Mott's case degeneration of this tract was not very intense, was more on one side, and was most distinct in the upper thoracic region. It extended downward to the eighth or ninth thoracic segment. The degeneration of the right pyramidal tract in my case was pronounced.

The lateral columns are not always diseased. Wladislaus Müller states that they are diseased in more than half the cases. Mott says a considerable number of cases show no degeneration or slight degeneration of the crossed pyramidal tracts. In the case of Friedreich's ataxia described by Lhermitte and Artom[27] the posterior columns of the cord were degenerated without degeneration of the lateral columns, except a marginal degeneration.

Mott found the lumbro-sacral ganglia much smaller than normal. The ganglia contained a number of myelinated fibers, and he detected atrophy or disappearance of many cells of these ganglia. I also have found degenerative changes in these ganglia.

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[26] Oppenheim, Lehrbuch.

Lhermitte and Artom[27] are the only authors I have found who refer to the radicular nerves. These nerves presented atrophy without the transverse neuritis described by Nageotte, and the condition was similar in my case.

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[27] Lhermitte and Artom, Bulletins et Mémoires de la Société Anatomique de Paris, 1907, p. 556.

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CONJUGATE DEVIATION OF THE HEAD AND EYES IN PARALYSING OR IRRITATIVE LESIONS OF THE CEREBELLUM.

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Read before the Philadelphia Neurological Society, March 25, 1910.

Conjugate deviation of the head and eyes resulting from a lesion of the cerebrum is believed to be away from the lesion in irritation and towards the lesion in paralysis, and the phrase, "The patient looks at the lesion," is generally accepted as correct for the deviation occurring in cerebral hemiplegia. The portion of the cerebrum directly concerned with conjugate deviation is not so well recognised, and this subject has received attention from Weisenburg. As regards pontile lesions the conjugate deviation is supposed to be inverse to that produced by cerebral lesions. As stated by Landouzy, a patient who turns his eyes towards the paralysed limbs on one side has a pontile lesion of a paralytic character; whereas a patient who turns his eyes away from his limbs of one side in convulsions has a pontile lesion of an irritative character. The evidence for the correctness of these statements is not so conclusive as that for the direction of the conjugate deviation in cerebral lesions, as conjugate deviation from a lesion in the pons is far more uncommon than from a lesion in the cerebrum.

The direction in which the head and eyes are turned from a lesion in the cerebellum is not determined satisfactorily, as conjugate deviation is usually the sign of a rapidly forming lesion, and is not likely to be persistent. Hæmorrhage or thrombosis of the cerebellum is rare. Starr says that in 187 consecutive cases of apoplexy examined post-mortem at the Presbyterian Hospital, hæmorrhage in or softening of the cerebellum was found in four cases only. He gives references to twenty-seven cases in the literature in which cerebellar hæmorrhage has been found after death.

When hæmorrhage occurs it almost always affects a considerable extent of the cerebellum, and even though it begins in a lateral lobe it is likely to extend into the vermis or fourth ventricle. It is difficult in cases of this extent to determine whether a hæmorrhage acts as an irritative or paralysing lesion. It is probable that a hæmorrhage near the surface of one lateral lobe, sparing the intrinsic cerebellar nuclei and therefore acting as an irritant, would produce a form of conjugate deviation inverse to that occasioned by a hæmorrhage destroying the intrinsic cerebellar nuclei and acting as a paralysing lesion.

Recent investigations concerning electrical irritation of the cerebellum are somewhat at variance. Von Bechterew states that irritation of one-half of the vermis in the dog causes a turning of the head, trunk and eyes towards the side irritated. It would appear from this that cortical irritation of the cerebellum is possible, but it is more probable that the irritation is accomplished through the intrinsic cerebellar nuclei. Horsley and Clarke have found that excitation of the surface of the cerebellum is without effect, but that when the insulated needles penetrate the cerebellar cortex a minimal stimulus gradually becomes maximal as the needle points are made to approach the intrinsic nuclei, which are highly excitable. The essential motor representations of the parts of the body in the intrinsic and paracerebellar nuclei are, in the former, the movements of the eyes and head; in the latter, more especially those of the trunk and limbs. The intrinsic nuclei are the nucleus dentatus, nucleus fastigii, and nucleus emboliformis vel globosus. The movement of the eyeballs from stimulation of the cerebellum is a steady swing over, which increases in contractile force as the excitation continues, and is without any clonic intermission of the motion or any development of any after-effect when the stimulation ceases. Direct excitation of the upper part of the nucleus dentatus alone evokes deviation of the eye of the same side, followed by conjugate deviation of both the eyes and head from the middle line to the same side.

Horsley and Clarke found that while excitation of the basal region of the nucleus dentatus with a maximal stimulus will produce, in addition to the homolateral conjugate deviation of the eyes and head, flexion of the homolateral elbow, a deeper excitation, *i.e.* of the paracerebellar nuclear region itself, produces widespread and additional effects, namely, extension of the contralateral elbow, hyperextension of the neck and trunk, with powerful extension of the lower limbs.

J. S. Risien Russell, in speaking of the ocular phenomena of cerebellar lesions, draws his conclusions chiefly from cases of tumour. He says: "As in cases of experimental ablation, defective power of movement of the eyes to the homolateral side, with insome cases even a slight tendency to deviation of the globes to the contralateral side in the resting condition, may be observed. It is, however, especially a difficulty in sustaining the movements of the globes to the homolateral side that is most evident. In this defect it commonly happens that the external rectus of the homolateral side shows a greater degree of disability than does the internal rectus of the contralateral eye. This is explained by the fact that whether or no defect of the conjugate movements is determined, the external rectus of the homolateral, and even that of the contralateral side to a less degree, gives evidence of weakness due to pressure effects on the sixth nerve. The external rectus paralysis is rarely complete, however, except when the tumour is so situated as to be able to exert pressure directly on the sixth nerve."

Russell refers to a case in which the symptoms clearly indicated a lesion of the right lateral lobe of the cerebellum. An abcesss was evacuated from this lobe. The patient lay curled up on his left side, with all his limbs flexed. His eyes were deviated to the left and presented lateral nystagmus. The right arm was markedly weak, while both lower limbs were slightly so. The right knee jerk was brisk and greater than the left.

It would seem to me that in this case a paralysing lesion was present, and therefore the deviation of the eyes was away from the lesion.

Russell finds that in the vast majority of cases vascular lesions in the cerebellum prove of pathological rather than of clinical interest, because death has resulted so rapidly or under such conditions as have not allowed of cerebellar symptoms being determined, or because the lesions have been of such a character that they could not be expected to cause a characteristic clinical picture. In hæmorrhage the patient usually dies rapidly owing to rupture of the blood into the fourth ventricle, or if living longer remains comatose, and does not permit of proper analysis of the symptoms in the way that might otherwise allow of information being obtained which might lead to a correct diagnosis.

Foci of softening in the cerebellum are usually so small as not to cause symptoms. This is to be accounted for by the free anastomosis of the cerebellar arteries, while the sources from which the cerebellum receives its blood supply make the occurrence of embolism altogether improbable.

Hæmorrhage is usually as much pontile as cerebellar. Russell refers to seven cases, but they throw little light on the direction of the conjugate deviation in paralysing as distinguished from irritative lesions of the cerebellum.

In the case reported by Laignel-Lavastine and Halbron the head and eyes, in an attack of apoplexy, were turned to the left. A hæmorrhage was found in the right cerebellar lobe; it implicated the nucleus dentatus and the vermis, and caused the formation of a clot on the right side of the floor of the fourth ventricle. Noteworthy in this case is the deviation of the head and eyes to the left from a lesion in the right lateral lobe of the cerebellum, and as the lesion was evidently a paralysing one, the case would support the view that in destructive cerebellar lesions the patient looks away from the lesion.

In Case VI., reported by Touche, conjugate deviation of the head and eyes existed toward the right. A large part of the left cerebellar lobe, and the left half of the posterior part of the vermis, were softened, and the softening extended to the nucleus dentatus. In Touche's Case V. conjugate deviation of the head and eyes was towards the left. Recent softening of the left cerebellar lobe was found implicating chiefly the cortex. From these two cases Touche concludes that recent superficial softening is accompanied by deviation of the head and trunk toward the lesion, while deep softening causes deviation of the head and trunk toward the opposite side.

Von Bechterew reported a case of hæmorrhage the size of a pea in the right lobus tonsillaris, resulting from a rupture of a branch of the inferior cerebellar artery. No statement is made regarding conjugate deviation of the head and eyes, and consciousness apparently was not lost. The case would have been valuable as regards the direction of conjugate deviation from cerebellar lesions, as would many others, had any observations regarding this matter in the onset of the symptoms been recorded.

I have recently had an opportunity to study a case which has important bearing on the subject of cerebellar conjugate deviation, and affords evidence that an irritative lesion of the cerebellum in man as well as in the dog causes turning of the head and eyes toward the lesion. It was one of small hæmorrhage in the left lateral lobe, which did not implicate the intrinsic nuclei of the cerebellum. Another hæmorrhage destroyed the right basal ganglia of the cerebrum, and there can be little doubt that so extensive a lesion as this was of a paralysing character, so far as conjugate deviation was concerned. Inasmuch as the head and eyes were turned to the left, the side of the irritating hæmorrhage in the cerebellum, and were turned away from the paralysing hæmorrhage of the cerebrum, the case shows that the symptoms produced by irritation of the intrinsic cerebellar nuclei predominated as regards conjugate deviation over those caused by a destructive lesion of the cerebrum.

It may appear singular that two hæmorrhages should have developed about the same time in the cerebrum and cerebellum, and that one should have been upon the right side and the other upon the left. The occurrence of hæmorrhage elsewhere in the brain in association with cerebellar hæmorrhage, however, is not unknown. Witte states that in 111 cases of cerebral hæmorrhage observed in the institute of Grafenberg during eight years the cerebellum was the seat of hæmorrhage in six, and in these six cases hæmorrhage elsewhere in the brain was present ; in five of them the hæmorrhage was in the central ganglia and the cerebral hemispheres. I have observed two recent hæmorrhages, entirely distinct from one another, in the basal nuclei of the cerebral hemispheres.

Had both lesions in my case been on the same side it would have been difficult to determine by which one the conjugate deviation of the head and eyes was produced. The deviation probably would have been toward the hæmorrhages, and would have been the combined result of the paralysing cerebral lesion and the irritative cerebellar lesion. It would be important to determine toward which side the conjugate deviation occurs when a destructive cerebral lesion is combined with a simultaneously developing destructive cerebellar lesion on the same side, *i.e.* one implicating the intrinsic cerebellar nuclei. Would it be toward the cerebral lesion, or away from the cerebellar lesion ?

The case to which I have referred is as follows :—I was asked to see a patient in Dr Laplace's service at the Philadelphia General Hospital on Feb. 9, 1910. He was a coloured man apparently about 70 years old, and had been brought into the hospital unconscious on Feb. 9, about 2.30 A.M. He had fallen on the street and was unconscious when picked up. Convulsions were not observed. The eyes and head were turned markedly to the left. The pupils were contracted and did not react to light. The left side of the face and left upper and lower limbs were paralysed as in ordinary hemiplegia. The arteries were sclerotic. The man had no control over the bladder and bowels, and was still unconscious. Both lower limbs were somewhat rigid. The patellar tendon reflex was exaggerated on each side, but Babinski's upward movement of the big toe was present only on the left side. The tendon reflexes of the left upper limb were exaggerated. Eighteen hours after the patient's entrance to the hospital, the intern recorded that the eyes looked forward with slight deviation of the right eye. This note would seem to imply that the left eyeball returned to a normal condition sooner than the right. It is uncertain, however, whether this slight deviation of the right eye was outward or inward, or whether deviation returned. Death occurred at 8 A.M., Feb. 10, 1910.

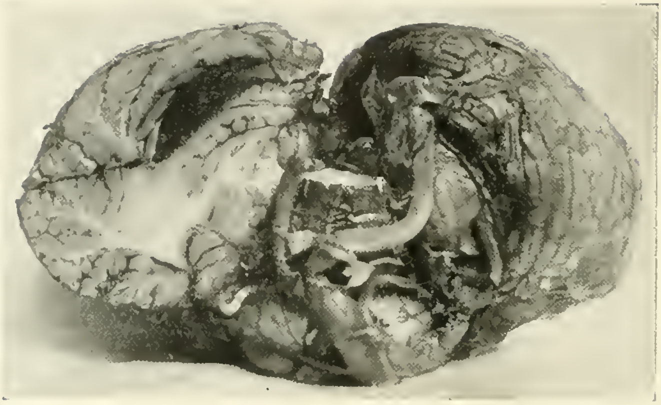

Fig. 1.

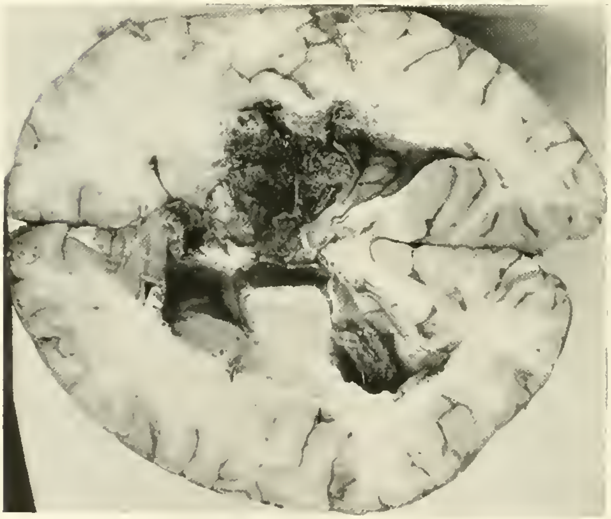

Fig. 2.

To illustrate Paper by Dr Spiller.

The deviation of the head and eyes toward the paralysed limbs without convulsions, away therefore from the cerebral lesion, was so unusual as to arouse my interest. A necropsy fortunately was obtained.

A recent hæmorrhage was found in the cortex and adjacent white matter of the left cerebellar lobe, and measured 2 cm. from the periphery of the lobe laterally toward the interior, and almost 2 cm. antero-posteriorly. It extended to within 2 mm. of the surface of the lobe. It was situated in the lower medial portion of the left lobe, and in no way implicated the intrinsic nuclei.

A recent cerebral hæmorrhage had destroyed the right optic thalamus, internal capsule and lenticular nucleus. It extended to the external capsule. The head of the caudate nucleus escaped.

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LEGENDS.

Fig. 1. Hæmorrhage in the left lateral lobe of the cerebellum. The deviation of the head and eyes was towards the left.

Fig. 2. Hæmorrhage in the basal ganglia of the right cerebral hemisphere.

SEROUS MENINGITIS.

By Samuel Leopold, M.D.

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It was not until the early part of the nineteenth century that different forms of meningitis became recognized. In Boenninghaus'[1] and in Schultze's[2] historical résumés we find that before this time there was much confusion as to the nature of these cerebral affections. Robert Whytt (1786) was probably the first physician to recognize the serious character in cerebral cases, in his descriptions of a case which he called *Hydrocephalus acuta*. Epidemic cerebrospinal meningitis attracted attention both in this country and abroad as early as 1806. The tubercular meningitis was not recognized until 1830. Finally, the progress made in distinguishing the various forms is well illustrated in the classification of Rilliet and Barthez[3] in 1855. They distinguished pathologically a simple or purulent meningitis from the tubercular, and clinically a convulsive form from a comatosed, etiologically a secondary and epidemic form.

Today, as the result of the advances in bacteriological studies as well as the microscopic, the varieties of meningitis are as numerous almost as the bacterial flora.

The serous form of meningitis has been recognized since 1846, when Dietl[4] called it the first stage of a severe purulent inflammation. Bouchout[5] gave it the name of *pseudomeningitis*, and distinguished it from the true by the absence of exudate and the presence of only congestion. Dupré[6] believed it to be functional, and we must thank him for the unfortunate term *meningism* which he gave to those conditions.

Confusion still existed as to the relation of serous meningitis and acute hydrocephalus until Hugenin emphasized their identity and Quincke,[7] in 1893, finally established it through lumbar puncture.

Serous meningitis may be defined as an inflammation of the pia arachnoid, of toxic origin, characterized grossly by a varying grade of fluid exudate and microscopically by a slight infiltration of round cells and edematous exudate.

The symptoms are similar to the other forms of meningitis, save that they are usually milder. Its course may be acute or chronic. Etiologically, a toxic origin is almost universally recognized. It has been attributed to other causes, however, such as ischemia, sunstroke, and hysteria. For some time the view had been prevalent among certain French writers that this condition was hysterical. Merry and Coureoux[8] record a case which gave the symptoms of meningitis, and with the idea of excluding this, they performed lumbar puncture. The fluid was removed and the operation gave instant relief.

In the variety with acute onset we find the history of some infection, such as typhoid, diphtheria, catarrhal enteritis, etc. Many cases have been reported in the literature in which the meningitis is associated with a pneumonia, scarlet fever, tonsilitis, rheumatism, erysipelas, measles, etc. In the case that I observed it occurred in association with a metritis of streptococcic origin. Middle-ear disease and nasal affections are frequently the primary areas of infection. Dana, in 1889, reported 24 cases following alcohol, inanition and prolonged use of narcotics.

From an analysis of many of the cases reported the clinical symptoms are as follows: The affection is seen in early age or youth. The onset may be sudden, but more often is slow. The fever, headache, and neck rigidity are usually present, but not of a severe grade. The temperature in the cases with slow or subacute onset rarely goes above 101.6°. Papillary hyperemia is almost always noted, and lumbar puncture and examination of the fluid shows either bacteriological or microscopic findings.

Symptoms of cerebral pressure or irritation may be noted, such as local palsies in the face and eye muscles (abducens paralysis, strabismus) and immobility or irregularity of the pupils. Delirium, stupor, and convulsions are also seen. The course is irregular, and its duration may extend from several days to several months.

Pathological studies have shown that two definite pictures in this condition are to be seen: First, the brain in which grossly no changes or only a congestion of the membranes, with varying degrees of serous exudate on its surface, but microscopically with a round-cell infiltration; and secondly, the form in which the ventricular cavities are markedly filled with fluid, and microscopically the changes in the tissues incident to pressure as well as to inflammation are noted.

In the first type we note a similarity to the cases described clinically by Quincke as acute serous meningitis with varying duration. This also corresponds to the forms described by Hugenin,[9] Schultze, occurring in the course of infections, and by a smaller group of observers as meningism. It is the form which resembles in its onset and in its symptomatology the picture of a purulent meningitis, and it is at times almost impossible to distinguish the two. Boenninghaus called it the malignant form of serous meningitis, but as Schultze rightly says, that is fallacious for the benign form of the former also terminates fatally.[10]

To this type belongs also the form described by Dana as *traumatic and alcoholic* serous meningitis, the so-called "wet brains," simulating closely a purulent meningitis.

Schultze and Hugenin and Arnold were probably the first to report cases in which they found grossly no changes, but found microscopically evidences of inflammation.

In a case illustrating this Schultze describes as follows: A young man, aged twenty-one years, with pain in the head, stiffness of the neck, sluggish pupils, with divergent axis, delirium, and coma; besides a croupous pneumonia a meningitis which showed itself in intense infiltration around the vessels, another illustration is seen in Hutinel's case.

I have had the opportunity of studying recently just such a case, following a septic endometritis in which the picture was that of a severe meningitis. At necropsy the brain and its membranes showed no changes, and microscopic studies showed only slight round-cell infiltration.

Case. I.—A. F., aged twenty years, was admitted to the obstetrical service of the University of Pennsylvania Hospital, October 4, 1905, with the diagnosis of pregnancy. Family history negative. Personal history, a suspicion of syphilis. On October 25, 1905, after the insertion of bougies to induce labor, the temperature began to gradually rise, without corresponding rise in pulse; twenty-four hours later the child was born. The second day the pulse went to 122 and the temperature to 103°. In addition the patient had severe chills and vomited. Vaginal examination showed tenderness in the cervical vault, and the broad ligaments were infiltrated. Blood examination showed, leucocytes, 29,000, and cultures of the blood showed the streptococcus. Seven days later the patient was delirious, had hyperesthesia over the entire body, with rigidity. Respiration became rapid; swallowing became difficult. November 3, 1905, or ten days later, the patient became stuporous; rigidity was more marked.

Examination by Dr. Spiller revealed lost knee-jerks and Achilles jerks; neck was painful on motion and stiff. No facial palsy. Eyegrounds negative. Temperature, 105.2°; respiration, 72.

The presence of rigidity of the neck, stupor, hyperesthesia, loss of tendon reflexes, and difficult deglutition, with fever, makes the diagnosis that of meningitis and streptococcic septicemia.

At necropsy, a suppurative metritis, necrotic endometritis, congestion of all the viscera, congestion and fatty degeneration of the kidneys. No gross changes in the brain.

Studies were made from the medulla, pons, midbrain, and cerebral cortex. Sections were stained with hemalum, Nissl, and Weigert stains.

No changes were noted in the meninges of the cortex or medulla. The sections of the pons and midbrain showed the mininges distended and containing an edematous exudate. The blood vessels in all of the sections were distended, and in some the vessels showed a collection of leukocytes along the wall, and in some cases a few leukocytes just out-side in the perivascular spaces.

The second pathological type corresponds to the benign serous meningitis of Boenninghaus, and the chronic serous meningitis of Quincke with chronic progress and at times acute exacerbations. This is the form which he says has been so frequently mistaken for brain tumor. He quotes the case of a worker, aged thirty-one years; duration of illness five months, slow progress, with headache, vertigo, rapid blindness, coma, optic neuritis, double abducens paralysis, disturbance of sensation on the right side of the face, facial paralysis; speech and deglutition difficult. Sudden death during dyspnea.

Necropsy showed all four ventricles dilated.

A case of Eichhorst's[11] illustrated how the chronic form may suddenly clinically simulate the acute. A student, aged twenty-three years, with an illness of three and one-half months' duration, began suddenly with chills, fever, headache, vomiting, and rigidity of neck.

High fever in first six days, afterward irregular. Pupils narrow, sluggish in reaction. Abducens paralysis on the left. No optic neuritis. Finally, sudden coma, irregular breathing, and after twelve hours death. At necropsy the entire brain was voluminous, convolutions flattened, all the ventricles very much dilated; ependyma thickened in places. The pia over the dorsal medulla thickened and adherent to the dura.

To correlate and explain the clinical phenomena and their relation to the pathological pictures in these cases would lead too far afield. Upon an analysis of all those factors which would cause cranial symptoms, one finds irritation of the cerebral centres by toxins, infiltration of the meninges by inflammatory products, mechanical increase in the size of the brain (emphasized more recently by Apelt[12]), or the increase in the quantity of the fluid present.

The diagnosis in some instances is only made at necropsy. Through lumbar puncture, study of the eye-grounds, previous history, and the course of the illness, a diagnosis should be reached in the majority of cases. The chronic form is sometimes taken for brain tumor. The acute form is usually confused with purulent meningitis. A comparison of the case reported above and the following case of purulent meningitis will emphasize their similarity.

Case II.—N. T., aged twenty-two years, entered the obstetrical service of the University of Pennsylvania on April 16, 1904. Prior to her delivery, on April 20, 1904, she complained of violent headache; after delivery temperature rose to 102° to 103°. Three days later she showed pronounced edema of the right eyelid; her headache still continued. Pelvic examination negative. Urine showed albumin. Heart, slight systolic murmur. Patient gradually went into a comatose state, and developed a left hemiplegia. She had several convulsions. The diagnosis of acute meningitis was based upon the headache, fever, left hemiplegia, and convulsions.

At necropsy the brain showed an extensive purulent exudate over the base and over the parietal lobe and in the ventricles.

The symptoms similar in these cases were the height of the temperatures, paralysis and psychic disturbances, delirium, and coma. The onset was more sudden in the purulent, the course more rapid, and headache intense. Convulsions and a left hemiplegia were also present.

A special form of the chronic serous meningitis has been recently described by Raymonde and Claude in the *Semaine Médicale* (1909, p. 577), to which they have given the name *circumscribed serous meningitis.* In the case which they describe, the patient, aged thirty-eight years, complained of fever and pain in his joints, headache, and general weakness extending over a period of several months. During this period he had diplopia, sleepy spells, trismus and difficulty in speaking, and convulsive seizures of the right side. There were no sensory disturbances, no ataxia, no visual disturbances. At operation a considerable amount of clear serous fluid escaped. The patient did not survive, owing to his marked weakness and malnutrition. Necropsy revealed a depression over the left Rolandic area, but no inflammatory or tubercular changes were to be noted. The collection of fluid had not been circumscribed, and no evidence of a well-defined cyst was seen. The fluid had occupied the subarachnoid space. Microscopic changes consisted of slight leukocytic infiltration, and the underlying cortex showed the effects of the serous exudate.

This form also may be mistaken for brain tumor or cerebral abcess, and operative interference offers a favorable prognosis. The presence of headache, torpor, rigidity of the neck, Kernig's sign, modification of the pulse, Jacksonian convulsions, and absence of papillitis and negative findings in the cerebrospinal fluid, should make its classification among the cerebral entities.

I wish to thank Dr. Spiller for the privilege of reporting these cases, and also Dr. Eva Rawlings for the privilege of examining the sections in the case of purulent meningitis.

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The Symptomatology and Pathology of Tumors of the Pituitary Body; Pathological Report of One Case.*

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(Read in the Section on Medicine, Medical Society of the State of Pennsylvania, Pittsburg Session, October 6, 1910.)

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Recent experience shows that tumors of the hypophysis may be successfully removed. It goes without saying that this is possible only when the tumor is comparatively small and, while an early diagnosis is desirable in any form of brain tumor, it is all the more important in tumors at the base of the brain because of the limited field of operation.

The diagnosis of tumors at the base of the brain in the region of the hypophysis is, in the beginning at least, difficult, as there are few characteristic or localizing symptoms. The text-books are not very clear, as a rule, in describing the symptoms of hypophysial tumor, the diagnosis of which is never very easy. A number of cases were collected from the literature with the view of studying anew the syndrome of tumors in this region in the hope of throwing some light upon the subject. It is regrettable that this study has not revealed many if any important facts which have not already been described by Roth and others.

The following case illustrates some of the difficulties to be met in arriving at a diagnosis of tumors of the pituitary body, and is interesting further in demonstrating how tumors of the brain may grow to considerable size and at the same time cause symptoms which are out of pr portion to the size and situation of the tumor.

W. S. was admitted to the Philadelphia Hospital on July 20, 1906. He was thirty-one years of age, a miner by occupation, and complained of loss of vision and headache. His father was an epileptic and his mother died of tuberculosis, otherwise his family history was negative.

Little could be ascertained of his previous history except that he had typhoid fever when nineteen years old. The patient further gave a history of recurrent headaches since childhood, but in the last six months the pain, which had been previously situated in the eyes, became occipital, occurred every morning, and was associated with nausea.

A year previous to admission there was a history of an attack, in which he fell to the ground without loss of consciousness. Six months later the attacks, which had recurred frequently, were characterized by transient weakness of the entire right side of the body. Later the right arm and leg became convulsed, and finally the entire body became involved. Sometimes the convulsions began in the legs and sometimes in the shoulders. There was never any loss of consciousness; he did not void his urine or feces during the attacks. He was completely blind a year ago but the exact onset of the failure of vision could not be obtained, though the loss of sight had been gradual.

On admission, the notes state that he lay in a semiconscious condition but answered questions in monosyllables, though the answers were not reliable. He was totally blind. Occasionally spasmodic contractures of the right leg were observed and fibrillary tremors were seen in the right quadriceps, the right pectoralis, and the left arm. There was no atrophy apparent, the man being of good muscular build. There was apparently no loss of power in the legs or arms and speech was normal.

Reflexes: The knee jerks were normal. Irritation of the plantar surface of the feet caused flexion of the toes. The cremasteric reflexes were preserved. The bicep jerks were normal and the jaw jerk prompt. The individual and coördinated eye movements were normal.

Three months later, October 2, '06, the notes state that the eyeballs were prominent and the tongue was protruded in the medium line. There was no incoördination, ataxia, nor astereognosis. No special enlargement of the hands, face or feet to suggest acromegaly was noted. The thyroid gland was not enlarged and supraclavicular swellings and infiltration of the skin was absent. The patient could not converge and there was no pupillary action of any kind obtainable.

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The features presented a full appearance, especially the cheeks, chin and throat. The eyelids were somewhat infiltrated as were also the cheeks, where the tissue felt exactly like typical myxedema. The trunk and limbs of the patient presented elsewhere the amount of subcutaneous adipose tissue usually seen in well-nourished individuals.

He sat all day in a chair with his head and shoulders bent forward, was even-tempered and required but little care. He appeared to be interested in what went on around him and talked rationally, although the answers were rather short and he rarely spoke of his own initiative. He walked normally when guided, the power in the limbs being good. The reflexes at this date were exaggerated but there was no Babinski phenomenon nor ankle or patellar clonus. There was no ataxia in the arms or legs. The right pupil was larger than the left but both were widely dilated and did not react to any test.

He was admitted to the Home for Incurables on August 6, '07, where he remained until his death, April 15, '08.

Examination made shortly before his death by myself resulted as follows: The patient was bedfast, the head somewhat retracted. He spoke only when addressed, and answered questions unreliably in monosyllables. There was complaint of constant temporal headache. It was practically impossible for him to walk more than a few steps as efforts to do so caused an attack in which the face became flushed, and there developed slight twitching of the muscles on the right side of the face and general rigidity of the whole body.

The grasp was equal and fairly vigorous. The knee jerks were increased on both sides and there was a tendency to ankle clonus on both sides. No Babinski phenomenon could be developed. He apparently felt the prick of the pin point distinctly all over the body. The eyeballs were prominent. There was no evidence of acromegaly, though the man was of large frame and there was more than the average amount of deposit of adipose tissue.

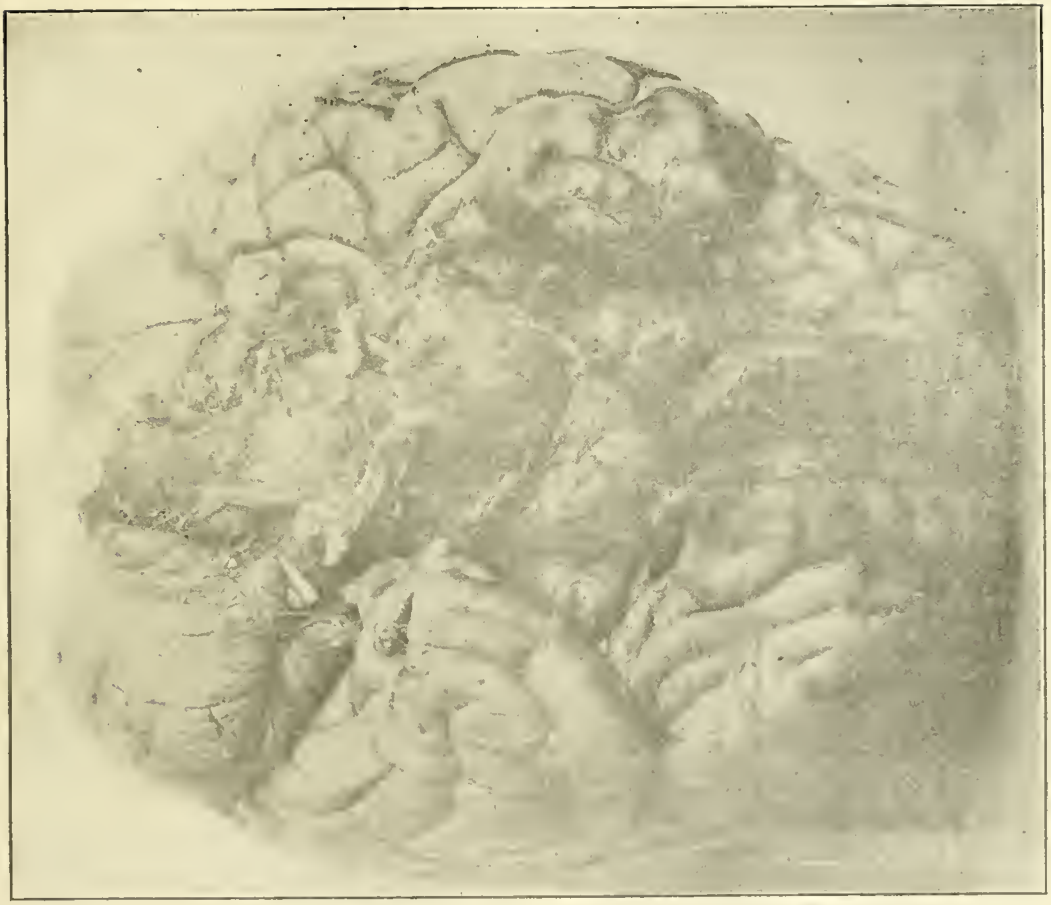
Examination of the eyes made by Dr. William Campbell Posey showed atrophy of the optic nerves which was not post-papillitic. A study of the intraocular muscles was impossible on account of the mental condition of the patient.

The autopsy revealed the presence of a large nodular tumor in the region of the pituitary body, measuring 6 cm. in the lateral diameter, 6 cm. anteroposteriorly and 7 cm. vertically. Imbedded in the basilar portion of the right temporal lobe was a second tumor, measuring 2.5 cm. by 1.5 cm. This was readily removed from its pocket; it had merely displaced the brain tissue without destroying it or becoming a part of it.

The first tumor described was a large nodular mass which had pushed its way upwards, so much so that the floor of the anterior horns of the lateral ventricles were displaced as high as the knee of the corpus callosum. This tumor was slightly adherent but could be readily separated from the base of the brain. The optic chiasm was stretched over the tumor downward to such an extent that it had become flattened out into a narrow band. The sella turcica was very much necrosed. Both cerebral peduncles were somewhat compressed, the right one being very much flattened out. The lateral ventricles were not materially distended and the brain substance itself was not anywhere invaded by the tumor. On section, the tumor was found to be smooth, slightly darker than the brain substance and was mottled by dark areas apparently due to hemorrhage into the substance of the tumor.

Sections were made from various parts of the tumor. Those from the posterior portion of the tumor showed a cellular aggregation of mononuclear cells, irregularly arranged and not enclosed in trabeculæ. The cells were large and the nuclei stained deep blue, the protoplasm pink. Sections from the middle portion of the tumor showed a somewhat different appearance in that the sections were traversed by blood vessels. In sections from the anterior part of the tumor the cells were surrounded by fibrous bands forming trabeculæ. Sections from other portions of the tumor showed this same arrangement, though in places the cells showed a disposition to infiltrate the stroma in a way which suggested the appearance of carcinoma. The cells had the appearance of the normal pituitary body.

Sections from the isolated tumor were stud-



Showing tumor in the region of the pituitary body. The optic chiasm appears as a narrow band.

ied by Professor A. J. Smith who kindly furnished me with the following report:—

The sections show over the surface a thin fibrous capsule, and internally it consisted of a delicate stroma bearing numerous comparatively thin-walled blood vessels. The stroma is covered by the essential cells of the tumor. In looser places there is a papilliform appearance, and in other places there is an appearance suggesting an adenocarcinoma. In still other fields there are large irregular spaces, filled with disintegrated blood. Fibrin and blood pigment are noted. In one portion the stroma is condensed into a delicate fibrillar and noncellularized matrix, the precise nature of which is uncertain.

The cells of the tumor are of the type of the glandular portion of the hypophysis, varying in shape from cuboidal to irregular forms due to varying pressure.

Each cell preserves a well-marked, slightly granular eosin-staining cytoplasm and a small round nucleus bearing considerable chromatin. There are no chromophilic cells. A diagnosis of adenocarcinoma was made.

Sections, made from the pons and the spinal cord and stained by the Weigert method and hematoxylon and acid fuchsin, showed nothing abnormal.

There was little in this case to point conclusively to a diagnosis of tumor of the hypophysis. Beyond blindness there was nothing characteristic, and localizing symptoms were absent. The blindness in itself was not indicative necessarily of tumor of the hypophysis, though the fact that the optic atrophy was not postneuritic indicated that there was pressure upon the optic tracts, chiasm or nerves. If this case had come under observation earlier before the blindness was complete, something might have been learned from a study of the fields of vision.

It is interesting to note that in spite of the pressure upon the cerebral peduncles, which was so great as to markedly flatten the one on the right, there was no paralysis and nothing in the symptomatology to suggest pressure there except the irregular convulsive attacks which were, however, more marked on the right side. It is further remarkable that a tumor which caused sufficient pressure to necrose bone, for the sella turcica was much necrosed, could exist without causing more pressure symptoms on the part of the nervous system.

A study of the symptomatology of tumor of the hypophysis was attempted from an examination of the reports of 169 cases, with autopsy, collected from the literature. Of 145 of these cases in which sex was mentioned 79 occurred in the female and 66 in the male sex.

The age of the patient was mentioned in 133 cases. More cases occurred between the ages of 30 and 40, there being 33. Between 40 and 50 years there were 23 cases reported; between 20 and 30 years there were 22 cases; between 60 and 70 years there were 18; between 50 and 60 years there were 16 cases; between 10 and 20 years there were 12 cases. There were 8 cases ranging from 2 to 9 years of age and 4 between 70 and 80 years.

Headache is a frequent symptom, occurring 78 times in 169 cases. In 5 of the remaining cases it was specifically mentioned as being absent and in the remaining 86 cases no mention was made. The situation of the pain is not characteristic. It may occur in all parts of the head, including the base of the skull. In two cases it is noted as being situated in the eye or eyeball. It may occur in the frontal, parietal, occipital or vertical region, or may be general. According to other observers headache occurred more frequently than these figures would indicate. In 38 cases collected by Roth,[1] headaches occurred in 81.8 per cent. In the statistics collected by Lebert,[2] headaches occurred in 85.7 per cent. Friedreich[3] found it present in 88.8 per cent., Ladame[4] in 71.4 per cent., Bernhardt[5] in 80 per cent. of the pure tumors of the hypophysis and in 70 per cent. of the tumors in the region of the hypophysis.

Convulsions, which occur in over 50 per cent. of cases of brain tumors, are less frequent in tumors of the hypophysis. In the 169 cases studied, general convulsions occurred in only 16 cases. There was stiffening of the body in 2 cases. In one case the convulsions were worse on the right side, in one there were convulsions of the left face, in one there was left unilateral convulsion, and in one there was convulsion of the eyelids and tongue. In Roth's cases there was history of epileptic convulsions in 12.1 per cent. but in 44 cases collected by Auche (quoted by Leclerc[6]) convulsions occurred 4 times only.

Vomiting, which is a common symptom of brain tumor, was mentioned in 36 cases out of 169. This is a contrast to the figures of Roth who found vomiting present in 42.4 per cent. of his cases.

Dizziness or vertigo was found in 16 cases or about 10 per cent. Roth's statistics gave 30.3 per cent. of the cases as suffering from vertigo.

The most frequent disturbance of consciousness was somnolence which occurred in 30 cases. In Soca's[7] case the patient slept for 7 months. Semicoma and stupor were noted in 17 cases.

There were only a few cases of mental symptoms not counting the cases of insanity. In 14 cases the mental condition was described as being either dull or stupid; 2, apathetic; 2, weakened or otherwise perverted.

Psychic depression and melancholia were found in 9 instances. Hallucinations were noted in 5 cases. The memory was lost or diminished in 15 cases. Childish manner, the Bernhardt symptom, was only noted in 1 case. Excitable or irritable temper was found in 6 cases.

Disturbance of speech, consisting of loss of speech, drawling speech, slow articulation or thick speech, was noted in 14 instances.

Disturbance of vision with headache is one of the most frequent symptoms, as well as the earliest. Vision was impaired or lost in 57 cases. Optic atrophy occurred in 17 cases, one of which showed neuritis in one eye and atrophy in the other. There were 7 cases of optic neuritis, two of which went on to atrophy, and 4 cases of choked disk. This is interesting in connection with the statistics of

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[1] Roth: *Arch. f. ophth.*, 1888, No. 34, p. 105.
[2] Lebert: In Roth.
[3] Friedreich: In Roth.
[4] Ladame: In Roth.
[5] Bernhardt: In Roth.
[6] Leclerc: *Rev. de. Méd.*, 1887, p. 977.

[7] Soca: *Nouv. Iconog. de la Salpétrière*, 1900, No. 13, p. 101.

Roth who stated that usually choked disk was not definitely noted and that, as a rule, the optic nerves were the seat of a primary atrophy. This is probably due, as Bernhardt believes, to the fact that the implication of the optic nerves is the result of direct pressure.

There were 9 cases in which the temporal fields on both sides were either contracted or showed a bitemporal hemianopsia. In 2 additional cases temporal hemianopsia was present on only one side and in 1 case there was left hemianopsia with loss of the color field on the right. In another case there was contraction in the right field of vision and in the temporal zone on the left. In 1 case the left lower quadrant of the temporal and nasal fields was lost. There were 4 cases in which the left temporal field was blind and the right eye blind, and in 2 cases the right temporal field was insensitive while the right eye was blind. In 4 other cases there was some disturbance of some parts of the fields of vision but imperfectly described. Sweeney[8] in a report of a case stated that homonomous hemianopsia of the nasal type may be the first manifestation of the abolition of sight.

The pupils are frequently dilated, there having been a history of this in 21 cases. The pupils were irregular in 4 cases, and failed to react to light in 13 cases. There was a history of immobile pupils in 4 cases. The pupils did not respond in accommodation in 4 cases, while in 1 case this failure was confined to the left eye. In 2 cases the pupils did not respond in convergence. The pupils were contracted in 3 cases. Third-nerve involvement was noted in 32 cases, causing ptosis, double vision or strabismus.

Ptosis was observed in 15 cases, being bilateral in 10, and confined to the left in 3 and to the right in one case. Strabismus in some form occurred in 17 cases.

Nystagmus was noted in 8 cases and photophobia twice.

Exophthalmos was present in 6 cases (Audry,[9] Leclerc, Pechkranz,[10] Battiscombe,[11] Dinkler,[12] and Thorburn[13]). This symptom was present to a moderate degree in my case.

Implication of the other cranial nerves is rare. There were only 7 cases in which the facial nerve was paralyzed. Sense of taste was lost in 2 cases and impaired in 8. Hearing was lost in 5 cases and diminished in 2. Sense of smell was lost or diminished in 5 cases. Tinnitis aurium was present in 4 cases.

In a few cases there was paralysis of the limbs. Hemiplegia occurred in 8 cases. In one instance the left arm was paralyzed, in another the left leg; in 2 cases both legs were paralyzed, in 1 case there was paralysis of the throat and in 1 case there was difficulty in swallowing.

Weakness of the legs and arms, however, was mentioned in quite a number of cases. Weakness of the arms was noted in 6 cases and weakness of the right arm in 1 case. There were, in addition to these, 9 cases in which the legs were weak. Weakness of the right leg was mentioned in 2 cases. General weakness was present in 24 cases and weakness of the right side in 2 cases.

There was rigidity of legs on both sides in 2 cases, on the left in 1, and general in 1 case. Rigidity of the limbs on both sides was present in 1 case and on the right side in 1 case.

Ataxia was found in a very few cases and staggering gait was noted in 9 cases.

In 9 cases there were pains in the limbs, hands or feet. In 9 cases there were pains in the body, in 4 cases pains were confined to the face and in 3 cases they were present in the neck.

Sensation was rarely disturbed. Paresthesia was complained of in the arms or body in 4 cases. In 1 case there was absolute anesthesia except in part of the face and forehead. In 1 case sensation was lost in the left arm and in another diminished around the nose. Pains in some part of the body were mentioned infrequently.

Excessive fat deposit was noted in 4 cases. Obesity as a symptom of tumor of the hypophysis has been recognized for a long time. Mohr[14] in 1841 called attention to it and Frölich[15] more recently cited a number of instances in which this symptom was described. Emaciation was noted in 6 instances.

The knee jerks were increased in 14 cases, quick in 4, absent in 5, lost on one side in 1, diminished in 3, irregular, being more active on one side than the other, in 3 cases. Ankle clonus was present on both sides in 1 case; on the left side in 1 case. Babinski's reflex was present on one side in 1 case.

Tremor was present in a few cases only and was noted as being present in the arm once, in the head once, in the tongue once, in the upper part of the arm once and twice in both legs.

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[8] Sweeney: *St. Paul Med. Jour.*, 1901, p. 81.

[9] Audry: *Lyon Méd.*, 1888, p. 73, Vol. 59.

[10] Pechkranz: *Neurol. Centralbl.*, 1899, p. 203.

[11] Battiscombe: *Lancet*, 1888, p. 970.

[12] Dinkler: *Deutsch. Zeitschr. f. Nervenh.*, 1891, 1, p. 371.

[13] Thorburn: *Lancet*, 1891, Vol. 2.

[14] Mohr: In Bregman and Steinhaus.

[15] Frölich: In Bregman and Steinhaus.

The thyroid gland was hypertrophied in 1 case, atrophied in 1 case and the right lobe was unduly prominent in 1 case.

Glycosuria was noted in 7 cases and polyuria in 4.

Insomnia was present in 5 cases.

Discharge from the nose was found in 2 cases (Johnston and Morrow[16] and Nettleship[17]). This symptom has been noted by Körner[18] who observed, in a case diagnosed as tumor of the hypophysis without autopsy, a clear watery fluid which came from the left nostril and he quoted 8 cases from the literature in which this symptom was present.

The character of the tumors was studied in 169 cases, including 2 in the pituitary region but not involving the pituitary body. Sarcoma was by far the most frequent form, of which there were 44 cases, including all forms of sarcoma. One of these was was either a sarcoma or a glioma. Next in frequency was the hypertrophied glands or hyperplasia of the pituitary body, of which there were 29 cases. Under a different head there were 14 cases described as adenoma and 17 as cystic; 8 cases were described as carcinoma and 2 as malignant. Epithelioma was noted in 8, endothelioma in 3. There were 8 cases of gumma and 4 of tuberculosis. Perithelioma was found in 1 case and psammoma in 1 case. Papilloma was present in 1 case, glandular tumor in 1, abscess in 1, fatty tumor in 1, glioma in 2 cases, lipoma in 1, fibrocartilagenous in 1, hemorrhage in 2 cases and in 22 cases the character of the tumor was not mentioned. It is worthy of mention that in some cases the tumor was discovered accidentally, no symptoms having been observed during life (Wolf,[19] Raymond,[20] Glaser[21]) nor any loss of vision (LeNoir[22]).

While it is not the purpose of this paper to discuss fully the question of acromegaly, since this symptom-complex occurs in a certain number of cases of tumor of the pituitary body, it is proper to refer here somewhat briefly to this subject.

Three theories exist to explain the occurrence of acromegaly: (1) That of lowered function of the pituitary gland (Marie); (2) that of increased function of the pituitary gland (Massalongo,[23] Tamburini[24] and Benda[25]); (3) and the view held by Gautier,[26] and later by Arnold,[27] Strümpel[28] and Vassali,[29] that acromegaly is due to a primary change in the general metabolism, the tumor of the hypophysis being a symptom and not its origin.

The whole subject is still in a state of confusion since it is to be noted that there are cases of tumors of the hypophysis without acromegaly, cases of acromegaly with tumor of the hypophysis and a few cases of acromegaly without tumor of the hypophysis.

The absence of acromegaly in cases of tumor of the hypophysis has been explained by the assumption that the gland has not been entirely destroyed (Burr and Riesman,[30] Walton and Cheyney[31]) and that the persistence of some of the glandular tissue is sufficient to maintain the function of the organ.

Kollarits[32] collected 53 cases of tumor of the hypophysis without acromegaly and came to the conclusion that tumor of the hypophysis was a part-symptom of acromegaly, while in my own studies of 169 cases of tumor acromegaly was present in only 52 cases.

Of these 52 cases of acromegaly the hypophysis was either the seat of a tumor or was hypertrophied in all cases except 3 (Virchow,[33] Israel,[34] Holden[35]).

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[16] Johnston and Morrow: *Glasgow Med. Jour.*, 1898, Vol. [illegible], p. [illegible].

[17] Nettleship: *Tr. Ophth. Soc. U. Kingdom*, 1884, Vol. 4, p. [illegible].

[18] Körner: *Zeitschr. f. Ohrenh.*, 1898, No. 33, 34, p. 1.

[19] Wolf: *Ziegler's Beitr.*, 1893, Vol. 13, p. 629.

[20] Raymond: *Bull. Soc. anat. de Paris*, 1888.

[21] Glaser: *Berl. klin. Wochenschr.*, 1893, p. 809.

[22] LeNoir: *Bull. Soc. anat. de Paris*, 1888, p. 1016.

[23] Massalongo: In Cagnetto.

[24] Tamburini: In Cagnetto.

[25] Benda: *Berl. klin. Wochenschr.*, 1900, p. 1205.

[26] Gautier: In Cagnetto.

[27] Arnold: In Cagnetto.

[28] Strümpel: *Deutsch. Zeitschr. f. Nervenh.*, 1897, Vol. 2, p. 51.

[29] Vassali: *Rev. neuol.*, 1903, No. 11, p. 560.

[30] Burr and Riesman: *Jour. Nerv. and Ment. Dis.*, 1899, p. 60.

[31] Walton and Cheyney: *Boston Med. and Surg. Jour.*, 1891, No. 141, p. 565.

[32] Kollarits: *Deutsch. Zeitschr. f. Nervenh.*, 1904, p. 88.

[33] Virchow: *Berl. klin. Wochenschr.*, 1900, p. 711.

[34] Israel: *Virchow's Arch.*, 1901, Vol. 164, p. 344.

[35] Holden: *Jahresbericht*, 1899, p. 870.

In this connection the studies of Schütte[36] are noteworthy. He found the hypophysis changed or hypertrophied in almost all of the 46 cases which he collected from the literature.

The most frequent forms of tumor found were sarcoma, of which there were 15 cases, and hypertrophy or hyperplasia, of which there were 20 cases. Cysts were found in 2, adenoma in 4, adenocarcinoma in 1, endothelioma in 1, glioma in 1, malignant tumor in 1, hemorrhage in 1.

Tamburini was the first to advance the theory of hypersecretion and he was later supported by Woods Hutchinson,[37] Gubler[38] and Benda who claimed that the extraordinary increase in the glandular cells as seen in three of his cases is proof of increased function.

Benda later described three forms of glandular cells in tumors of the hypophysis which he believed were intermediary forms merging one into the other, all three forms showing a nucleus rich in chromatin and with glandular protoplasm. He believed that the chromophilic cell in the normal as well as the pathological hypophysis appeared as the same element.

Fraenkel, Stadelmann and Benda[39] in 1891 called attention to the fact that the Benda type of cell resembled that described as occurring in hyperplastic conditions as well as those described as found in sarcoma or angiosarcoma. He quotes Gubler who examined the hypophysial tumor in so-called acute malignant acromegaly and is convinced that not only his tumor but those also described in six other cases are instances of stroma. They also cited the cases of Claus and Van der Stricht,[49] Woods Hutchinson, Marie and Marinesco[41] in which the cells resembled the normal hypophysis.

Later Vassali in 1903 claimed that acromegaly was a disease of nutrition in which the cause was unknown and in which the increase in volume of the pituitary body is one of its consequences, the tumor being a secondary phenomenon. The tumor of the hypophysis is always a simple hypertrophy characterized by the presence of chromophilic cells in abundance, indicating functional activity.

Cagnetto[42] studied the question of the relation of the chromophilic cells to acromegaly and found them present rarely in one case, numerous in the second case, sparsely in the third case, in masses in the fourth case and in a case of tumor without acromegaly. He believed that he had established an argument against the hypophysary theory, especially the one which claims an increased function of the pituitary body. He shows also that acromegaly occurred without hyperplasia of the hypophysis, that it occurred with tumor of the hypophysis which originated from functionating elements, and, finally, that it is possible to have a tumor of the hypophysis with numerous functionating cells without acromegaly. Hyperplasia of the chromophilic cells was also described by Lewis. On the other hand, however, Zak and Miller did not find them present in their cases.

Zak[43] called attention to the fact that it was difficult in the literature to define the difference between adenomata and sarcomata and emphasized the importance of the use of the modern technic, especially that suggested by Benda, to elucidate this question.

What part the hypophysis plays in the development of acromegaly must at present be still further a matter of doubt, in view of the fact that in Israel's case, no increase of the chromophilic cells nor enlargement of the hypophysis was seen and that in the cases of Virchow, Israel, and Holden no tumor whatever was found.

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[36] Schütte: *Centralbl. f. allg. Path.*, 1898, p. 591.

[37] Woods Hutchinson: *N. Y. Med. Jour.*, 1900, p. 134.

[38] Gubler: *Rev. Neurol.*, 1901, p. 547.

[39] Fraenkel, Stadelmann and Benda: *Deutsch. med. Wochenschr.*, 1901, No. 27, p. 513.

[40] Claus and Van der Stricht: *Ann. de la Soc. de Méd. de Gand.*, 1893, No. 72, p. 71.

[41] Marie and Marinesco: *Arch. de Méd. expér.*, 1891, p. 539.

[42] Cagnetto: *Virchow's Arch.*, 1907, p. 197.

[43] Zak: *Wien. klin. Rundschau.*, 1904, p. 165.

In connection with the theory of hypersecretion it is interesting to note the relation which exists between the thyroid gland and the pituitary body. Comte[44] claimed that the hypophysis played a vicarious rôle in cases in which the thyroid function was embarrassed by a pathological process and Marie and Marinesco saw functional compensatory hypertrophy of the hypophysis in rabbits in which the thyroid body had been removed.

Rogowitsch's[45] experiments in 1889 on guinea pigs proved, he believed, that there was increased activity of the pituitary body after removal of the thyroid gland, and Stieda,[46] as the result of experiments on dogs, looked upon the hypertrophy of the hypophysis after the removal of the thyroid gland as an expression of increased activity of the hypophysial tissue. Hofmeister[47] also caused hypertrophy of the glandular part of the hypophysis in young animals by removal of the thyroid gland.

An effort was made to study the effect upon the growth of animals by Caselli[48] who claims to have arrested development in dogs after extirpating the epithelial part of the hypophysis. Friedmann[49] however was unable to cause any change in the development of young cats by removing the hypophysis.

The general symptoms of intracranial growth, namely, headache, convulsions, mental impairment, optic neuritis or atrophy, vomiting and vertigo, occur to more or less extent in cases of tumor in the region of the hypophysis.

Headache, which occurs in the majority of cases of intracranial growth, is less frequent than usual in tumors at the base, if the figures in my collection of cases can be trusted. Convulsions are less frequent in tumor of the hypophysis than in other situations. Vomiting is not frequent though present in a fair number of cases. Mental impairment, somnolence or disturbance of consciousness is a moderately well-marked symptom, while vertigo is less frequent than when the tumor involves the frontal or occipital regions.

Disturbance of vision is a frequent and fairly constant symptom, though in the cases studied only 57 of the 169 cases showed impaired vision. The loss of vision in these cases is due, in most instances at least, to pressure upon the chiasm causing optic atrophy. Choked disk is uncommon, having been noted in only 4 cases. Changes in the field of vision were noted in 24 cases. Bitemporal hemianopsia is probably the most characteristic of the symptoms of tumor of the hypophysis, and can be looked upon as practically the only localizing symptom.

Palsy of the limbs occurs but infrequently and when present gives no definite information.

Exophthalmos was noted in only a few cases, but probably is more frequent than the statistics would indicate.

Obesity, acromegaly, gigantism are symptoms of note. In this connection it is of interest to quote Brissaud[50] who concluded that, if the overgrowth occurs in childhood, gigantism and not acromegaly occurs; if in adult life it results in acromegaly.

The use of the *x*-ray in the diagnosis of tumors of the hypophysis is of much value in arriving at a diagnosis in some of these cases. Some distortion of the sella turcica may be found.

In conclusion I wish to emphasize the importance, in making a diagnosis, of early studies of the fields of vision.

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[44] Comte: Ziegler's *Beitr. z. path. Anat.*, XXIII., p. 90.

[45] Rogowitsch: Ziegler's *Beitr.*, 1889, Vol. 4, p. 455.

[46] Stieda: Ziegler's *Beitr.*, 1890, Vol. 7, p. 532.

[47] Hofmeister: *Beitr. zur klin. Chir.*, 1894.

[48] Caselli: *Rev. Neual.*, 1900, p. 905.

[49] Friedmann: *Jahresb. f. Neurol. Psychiat.*, 1902, p. 135.

[50] Brissaud: In Bregman and Steinhaus, *Jour. de Neurol.*, 1907, p. 301.

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November, 1909.

THE CUSTOM OF DELIVERING INAUGURAL ADDRESSES, WITH SOME DISCUSSION OF THE METHODS OF UNDERGRADUATE TEACHING IN DIFFERENT COUNTRIES.[1]

By Charles K. Mills, M.D.,
Professor of Neurology in the University of Pennsylvania.

This evening begins the one hundred and forty-fourth session of the medical school of the University of Pennsylvania. Its sesquicentennial will be celebrated a short six years hence, a celebration which will, I have no doubt, be a worthy one, recalling the glories of the past as illustrated both by the men who have served our university, and the results which have been accomplished by their service. During the years which have already elapsed more than thirteen thousand graduates have left our medical halls to use the knowledge and skill there acquired for the benefit of their fellows.

The custom of delivering lectures introductory to the medical course is as old as the history of the school, and few gaps occurred in the record of these lectures until eleven years ago, when they were discontinued because of some difference of opinion as to their value; also because it was thought better to have the opening of the medical school take place in conjunction with that of some departments of the University. They were not resumed until the opening in 1904, of the new medical hall or laboratories building in which we are now assembled. Some good has come out of a custom one of whose purposes is to bring the entire student body together at the beginning of the college year.

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[1] Address to the General Medical Class at the Opening Exercises of the One Hundred and Forty-fourth Session of the Medical School of the University of Pennsylvania. Delivered in the Amphitheatre of the Medical Laboratories Building, September 24, 1909.

It was not only customary in days not far removed to give introductory lectures, attended with more formality and ceremony than at present, to the general medical course, but the professor at the head of each department considered it his duty to open his particular course with some form of inaugural address. Some of these addresses have been collected into volumes, one of the most interesting of these, published in 1801, being a series of six by Benjamin Rush to courses of lectures upon the institutes and practice of medicine.

Chapman and Jackson apparently found especial pleasure in preparing and putting in print their inaugural addresses, in the delivery of which they took the opportunity of exhibiting those graces of rhetoric and oratory for which they were both noted.

A glimpse at some of these lectures somewhat after the manner of the *Review of Reviews* might prove instructive and possibly of interest, recalling some of the history and best traditions of our medical school. In the library of the University and in that of the College of Physicians of Philadelphia are printed introductory addresses sufficient in themselves to make several bulky volumes. These addresses, with those delivered at other medical schools, constitute a literature of their own. In not a few instances they serve to fix attention upon great periods in the history not only of the University of Pennsylvania, but of medical education in this country.

In the pioneer period, in the list of inaugural speakers are found the names of Morgan, Shippen, and Rush; in later periods, those of Wistar, Chapman, Jackson, Gibson, Hare, George B. Wood, and Joseph Carson; and their successors, in a new period of university advance, Alfred Stillé, Agnew, Pepper, and Horatio C. Wood. These are names without which the history of American medicine cannot be written. Within these walls, in the few years of the existence of this great laboratory and teaching building, addresses worthy of perpetuation have been given by some of those who will be your teachers.

Time-honored topics, philosophical and ethical, the conjunction in medicine of reason and observation, of science and art, the responsibility of physicians, autobiographical memoranda, and glimpses of the personalities of those who taught the speaker, the life of some great medical leader like Sydenham, the story of the school's beginning and prophecies of its expansion, the exploitation of Philadelphia as a medical centre, criticism of medical education in the present, and forecasts for its future—these are some of the subjects of discourse in the two score or more inaugural lectures which I have examined.

The conduct, actual or assumed, of the student, whom we can imagine sometimes listening like the obsessed parishioner to the warning priest, is a not infrequent topic. Just how it was with the student of the old days I am not altogèther prepared to say, but evidently the worthy professor into whose hands he was about to fall had full belief in the doctrine of original sin, and the necessity of counteracting its effects by goodly counsel. In those days the student was not furnished with the healthy outlets to youthful virility and energy which are now so amply supplied by societies and fraternities, by students' halls, by well-stocked libraries, by supervised physical exercise, and athletic competition.

The glimpses we obtain of the men of one generation through the sketches or light touches of a succeeding generation who were their students have always a special charm, and thus have been handed down from the beginning of the school personal characteristics which would otherwise be lost. Thus appear before us Rush, ruddy and radiant even in wrinkled old age; the fiery tones and resolute face of Barton; Physick, the father of American surgery, frail of body, wise, grave, and impressive; Wistar, learned and courtly, the idol of his classes; handsome young Dorsey, who in his brief career acquired the name of the American Sir Astley; and James, orderly, scholarly, and dignified.

The method of personal recall in students' addresses is worthy of commendation. Pope tells us that the proper study of mankind is man. One of the most inspiring studies for mankind is men—men who have bravely and worthily lived their lives.

Dr. Hobart A. Hare, in an address delivered by him before the second annual meeting of the undergraduate medical society of the University of Pennsylvania, in the spring of the present year, on "The Faculty of the Early Eighties and its Influence on Medical Men," has given us a pleasant and inspiring picture of Stillé, Agnew, Leidy, Goodell, Ashhurst, Wormley, William Pepper, Penrose, Harrison Allen, H. C. Wood, and James Tyson.

It is well that such personal impressions should be handed down. They are encouraging and inspiring to the man who loves his profession and his school.

Permanent memorials to medical men are in our country few in number. The monuments and tablets which tell of their achievements can almost be counted upon the fingers of the two hands—one to Rush and another to Gross, in Washington; one to Morton, in Hartford; to Maguire, in Richmond; to Leidy, on our own city hall plaza; to Dr. Pepper, on the University campus; a tablet to Oliver Wendell Holmes, in King's Chapel, and a few other memorials here and there are all of which we can boast.

It is not so in Germany, where almost every university town has its monument, or memorial to some great medical man, some one who honored the university as a teacher, and was regarded as an ornament to his city and the state. The walls of the dwellings and college buildings in many university towns are largely covered with tablets which tell that this or that medical man honored this room or this house by living in it. Even the German hackman seems to have caught the sentiment of regard for professional and scientific worth and is rather more likely to take you to the statue or to the old abiding place of Herr Professor Doctor than to the monument or the buildings which tell of battles fought and victories won.

In France the monuments and other permanent memorials to great physicians and surgeons are comparatively numerous. These memorials sometimes take the form of hospitals named after distinguished members of the profession—in Paris of such men as Bichat, Louis, Laennec, Trousseau, Charcot, and many more. In the little city of Tours, in the department of Indre et Loire, of the old province of Touraine, is a monument, with three life-sized medalions, to Bretonneau, the discoverer of diphtheria, Velpeau, the great French surgeon, and Trousseau, the renowned clinician. These three illustrious members of our profession were natives of the department.

If we have not memorials like those of Germany and France, in brass and bronze and stone, we can at least keep alive the traditions of the great men who have preceded us.

Now and then one of the introductory lectures had some special quality of interest. The manner in which the professor in the earlier years of our history acquainted the class with his qualifications for the position seemed to assume an entire ignorance on their part of the history of the new incumbent. Gibson, in his lecture introductory to his first course on surgery, tells, in his complaisant way, his own personal story, not forgetting the boarding-house mistress to whom he confided the fact that, after hearing Dr. Physick, he had made up his mind some day to succeed to his position.

Gibson seems, like Napoleon, to have had confidence in his star, and weaves much of the record of his life around his early prophecy.

Referring to the fact that Americans have been charged with talking too much about themselves, Gibson does not hesitate to admit that he sees no objection to doing this. In this connection he tells a story of Sir Astley Cooper, under whom he learned some of his surgery while in London. Sir Astley said to him, "Why is it that all you Americans are so fond of using the word I, or of speaking of yourselves?" "Because, my good Sir Astley," he replied, "we are descendants of Englishmen, and for the same reason that you, for the last two hours, have been talking so continually of yourself, so much to my own edification and advantage."

For a humor which was not intended, the introductory lecture before the general class delivered by Dr. Gibson on November 4, 1844, is unique. In this lecture he paints in glowing colors the excellencies of Philadelphia, its freedom from noise and bustle, its clean streets, its spacious squares, its buoyant atmosphere, where consumption is almost unknown, and where a refined and cultivated literary and moral society has exercised a salutary influence on the rising generations, Philadelphia's religious character and observances, its missionary zeal, and its Sunday schools. He enters a defence of the city against the accusations which had grown out of the recent native American riots.

Fearing that the glowing picture of the sobriety, refinement, and particularly the salubrity of Philadelphia might awaken in his hearers the idea that disease, the study of which was their particular quest, could not exist under such favorable influences and conditions, Gibson throws an anchor to windward by telling them that the country surrounding Philadelphia is not free from malarious and other pestiferous influences which serve to fill the hospitals and afford opportunities for clinical teaching of varied and practical character. His hearers are encouraged in similar ways with regard to accidents.

The writings of George B. Wood have a charm which one cannot help but feel, whether it be in reading his general addresses, his descriptions of disease, or his discussion of the nature and use of drugs. In all that he wrote he showed the same perspicuity, smoothness of diction, and controlled enthusiasm. In the introductory lecture to the course delivered in 1846, Dr. Wood paints in a manner that could not be excelled the ideal physician, with an appeal to his audience to fulfil the model presented. He could not conceive of a good doctor who was not conscientious, and he dwells upon the relations which he should have to his professional brethren, holding these as second only to those which he has to his patients.

Among the inaugural addresses in the University of Pennsylvania are some of historical value, four of these being those of John Morgan in 1765, of Benjamin Rush in 1791, of Joseph Carson in 1865, and of Alfred Stillé in 1874.

In a strict sense the address of Morgan was not an introductory lecture, although it marked the beginning of the medical school. It was delivered at a public anniversary commencement of the College of Philadelphia in 1765. Morgan had just been appointed to the professorship of the theory and practice of medicine, the first chair in the school of which he and William Shippen, Jr., are justly regarded as the founders. Morgan advocated the practice of medicine in America along the lines pursued in Edinburgh and other cities abroad.

In the last part of his lecture he considers at some length the establishment of a medical school in America, referring to the great advantages of bringing students together, of those which accrue to the city and state in which such a school is placed, to those which would spring in a country like America from the opportunities offered to make discoveries in natural history. He makes a strong appeal to the ambition, industry, and enthusiasm of the students, gives warm thanks and high appreciation to the work of the trustees, and does not forget the good monarch across the seas who has recognized the Philadelphia seminary. As was becoming in one delivering so scholarly an address, he closes with a quotation from Quintus Horatius Flaccus. The commencement exercises and the delivery of this address were distributed over two days. It is difficult for us to conceive of a modern commemoration audience listening with enthusiastic interest to so long a discourse, however able. In this lecture Morgan struck a prophetic note, forecasting the development of other institutions for like purposes, and the organization of societies for the promotion of medical knowledge out of the establishment of the new school.

The introductory lecture delivered by Dr. Rush in 1791 marked one of the great periods in the history of the University of Pennsylvania. The College of Philadelphia, during the troublous times of the Revolution, became much disorganized, and medical teaching went on in an irregular and indifferent manner. In 1779 the charter of the college was abrogated and its property transferred to a new institution which was called the University of the State of Pennsylvania. Some medical instruction was given under the auspices of this new institution, most of the professors of the college, however, declining for a time, and some of them altogether, to take any part with the new university. In 1789, largely through the influence of Franklin, the charter and privileges of the college were restored, and from that time until 1791 the city had two medical departments or colleges, neither of them doing its work very effectively. One of the members of the old college Faculty, Dr. William Shippen, acted as professor in both institutions. In 1791 the two institutions were reunited under a new charter with the name of the University of Pennsylvania. It was on the occasion of the opening of the medical school, after this reunion, that Dr. Rush delivered his introductory lecture.

Dr. Rush first congratulates his class on the union of the two medical schools of Philadelphia, by which union the ancient harmony of the different professors of medicine will be restored. For the first time, he asserts, by the distribution of the various branches of medicine into so many professorships, an equality can be claimed with the oldest universities in Europe. Although the expenses of a medical education in America will be increased, these will not be proportionate to the advantages gained, and will still be much less than those necessary to obtain a medical education by going to foreign schools. Rush emphasizes two things in comparing a medical education received at home with that obtained by going abroad, first, that the student here studies the diseases of the country in which he will later practice, and in the second place he will be able to deviate from the methods taught abroad to one based upon principles derived from the study of diseases in his own country.

Rush then directs special attention to the duties assigned to him in the course—those of teaching the institutes of medicine and illustrating the subjects taught by such diseases as occur in the Pennsylvania Hospital during the winter.

After these preliminaries his lecture concerns itself with a discussion of the necessary connection between observation and reasoning in medicine. The advantages of empirical and theoretical methods are discussed, and the necessity both of experience and of theory properly adjusted to each other is shown. The lecture, besides being interesting historically, is full of thought, one of the ablest inaugural addresses which fortunately have been preserved. Rush directs attention to the systems of Stahl, of Boerhaave, Cullen, and Brown, showing the values as well as the deficiencies of each. He indicates that he will follow these great men in so far as he believes them to be right, advancing at times views of his own, the result of personal experience, but speaks of his willingness to revise these views and acknowledge them as erroneous when corrected by further experience and study.

It may be of some interest to the students of today to say a few words regarding the terminology employed by our forerunners in describing the subjects upon which they lectured. For many years, a century and more in fact, the term institutes of medicine was in frequent use. The six courses of lectures which were inaugurated by the lectures included in the little volume of Rush to which I have referred, were designated as courses upon the institutes and practice of medicine. The professorship of the institutes of medicine was long one of the chairs in this university and in other medical schools, and in a few the designation is still used, or was until a very recent period. By the institutes of medicine, as the term was most frequently employed, was to be understood the basic scientific subjects, or some of them, on which medicine as a whole rested, or from which a knowledge of it was in large part derived. As thus employed it was meant to describe the most important of the theoretical aspects of medicine, and Rush himself, in the lecture here referred to, describes the institutes of medicine as including physiology, pathology, and therapeutics. As time went on, the subjects discussed by the chair of institutes were more and more limited, until physiology was practically the only one left, pathology and therapeutics being relegated to special professorships or included in some other combinations, as, for instance, that of materia medica, pharmacy, and therapeutics. In my own days as a student, and for many years after my entrance into the medical school, the professor of the institutes of medicine confined his teaching almost entirely to physiology. And so it has come to pass that the more appropriate method has been adopted of calling the chair itself that of physiology. Both pathology and therapeutics, comparatively circumscribed in the days of Rush, have enormously expanded, so that, as you will learn in the present course, it has become necessary to subdivide the teaching of both in such a way that different branches of these subdivisions of the old chair of institutes are now delegated to various teachers.

Probably the most important date in the history of our medical school, after the union in 1791 of the medical college of Philadelphia and the University of the State of Pennsylvania, under the new title of the University of Pennsylvania, was the year 1829, the time of the opening of the then new building for the medical department on Ninth street near Chestnut.

The faculty of the school then consisted of a group of the greatest men who have honored the university as teachers—Physick, Chapman, Gibson, Coxe, Hare, James, Horner, Dewees, and Jackson—the last three not holding full professorial chairs, although destined soon to become their occupants.

For a few years the academic department had been like a pigmy appendix to the medical colossus. The number of students in the college was as few as twenty-one. The college department had, with the medical school, been removed to the house on the square between Market and Chestnut and Ninth and Tenth Streets, which had been built in 1800 for the accommodation of the president of the republic, and after the removal of the seat of government to Washington had been transferred to the trustees of the University of Pennsylvania. The medical department continued to occupy this house, which, however, was not suited to its purposes, while the college department had returned to its old quarters on Fourth Street. The Presidential mansion was razed and two plain but substantial and commodious buildings were erected in its place. In one of these, that on the Chestnut Street side, the medical department was installed in 1829; in the other, toward Market Street, the collegiate department, in 1830.

The college Faculty was reorganized with the Rev. William H. DeLancey at its head, and with new life infused, rapidly increased the number of its students so that on September 30, 1830, there were as many as 126.

The corner stone of the new medical hall was laid March 21, 1829. At the time of this ceremony the degree of Doctor of Medicine was conferred on 107 students, and after the exercises an address was delivered by the Provost. In the succeeding autumn the building was ready for occupancy, and the session of the medical school was opened on November 2, 1829. In the minutes of the Faculty of the University of Pennsylvania for October 17, 1829, this entry was made:

"The following order of introductory lectures was agreed upon:

Dr. Chapman, Monday, November 2, 12 M.
Dr. James, Monday, November 2, 1 P.M.
Dr. Hare, Tuesday, November 3, 12 M.
Dr. Coxe, Tuesday, November 3, 1 P.M.
Dr. Gibson, Wednesday, November 4, 12 M.
Dr. Horner, Wednesday, November 4, 1 P.M.

"Dr. Physick declined opening the course on anatomy, and the exchange was made by mutual consent of Drs. Chapman and Horner."

This statement about Dr. Physick, which may at first sight not appear intelligible, evidently means that as Dr. Physick was the oldest member of the faculty, it would have fallen to him by right of seniority to give the first of the introductory lectures on Monday. Dr. Chapman, next in seniority, took his place, and Dr Horner, then assistant to the chair of anatomy, gave the introductory to the anatomical course, and being a junior was properly placed at the end of the series. In 1831 Dr. Horner succeeded Dr. Physick as professor of anatomy.

It will be seen from the above minute that the delivery of an introductory address to each of the courses in the medical department was considered so important by the trustees that formal selection was made of the orator and of the time of delivery. Apparently these lectures introductory to the separate courses of instruction took the place of a general introductory. The students of the entire medical school doubtless were expected to attend the various introductories, the regular courses of instruction not beginning until after their delivery. Dr. Chapman should be regarded as opening the first session in the new medical hall, as his lecture was the first on the list. I have not found a printed copy of this lecture, although many of Dr. Chapman's introductory addresses were published in the years which soon followed.

The medical department, free to expand in its new quarters, added new honors to those already achieved. Many of the most interesting and important of the introductory addresses which have been preserved were delivered during the two decades beginning about 1830.

An introductory lecture of importance, not only in itself, but because of what grew out of it, was that delivered by Dr. Joseph Carson at the opening of the medical school in 1865, the centenary of the founding of the school. In this lecture Dr. Carson discussed the events preceding and attending the foundation of the medical department, also giving some account of the lives and characters of the men interested in its foundation and its early history. This lecture was not published as delivered, but out of it grew the first systematic History of the Medical Department of the University of Pennsylvania, from its foundation to the year of the publication of the volume, 1869.

Another notable introductory lecture and one which remains as a part of the best historical literature of the University of Pennsylvania, was that delivered by Dr. Alfred Stillé at the beginning of the one hundred and ninth session of the medical department of the University of Pennsylvania, October 11, 1874. This was at the time of the opening of the first session of the school after the removal of the University from Ninth Street to the present campus. I was present at this lecture, and well remember the occasion and the effect of the address upon the audience of students, professors, alumni, and invited guests, which crowded the great amphitheatre. It was a day of enthusiasm, and one which spoke well for the custom of delivering inaugural addresses.

The history of the removal of the University from Ninth Street to West Philadelphia; the rules which should govern us in the pursuit of medical knowledge; the similarities and differences between science and art; and prophecies of advance for the school in the early future were among the main topics of the address, which was scholarly, dignified, and impressive. It was not long after the delivery of this lecture that the old two years' régime gave way to a three years' course (1877). The voluntary fourth year course was instituted in 1881–82, and beginning with the session of 1893–94, four years were required. The time is not far distant when five years will be required, as in Germany, France, and England.

Most interesting to anyone concerned with the history of the medical department of the University of Pennsylvania is a comparison of the conditions and circumstances of the school at the periods of the four great introductory lectures to which I have especially referred. In 1765 there were but two professors in the medical department, Morgan and Shippen, and it was not until 1768 that the school furnished any graduates, and these only ten in number, who received the degree of bachelor of medicine, or bachelor of physic, as it was then commonly called. This was the only medical degree in course conferred by the University for twenty-one years, although the degree of doctor of medicine, on the completion of certain requirements, was sometimes given one or two years after the reception of the bachelor's degree. The number of graduates in the separate schools in 1791, at the conclusion of the session, before the delivery of Dr. Rush's address, was, for the college of Philadelphia five, and for the University of the State of Pennsylvaina, one. The number of graduates, of the united medical schools, at the end of the session of 1791–92 was six.

After the union of the College of Philadelphia and the University the teaching of medicine in Philadelphia steadily advanced, and by the beginning of the nineteenth century this city was universally recognized as the great American medical centre. The medical classes increased with rapidity. Dr. Meigs, in an introductory lecture to the class in obstetrics at the Jefferson Medical College in 1841, speaks of Rush as surrounded by five hundred students during the delivery of his lectures in 1809. The number of main professorial chairs was increased to seven early in the century, and was continued at this number until about 1870. These chairs were anatomy, chemistry, institutes of medicine (physiology chiefly), materia medica and pharmacy, medicine, surgery, and obstetrics.

In 1829, when the medical hall on Ninth Street was opened, the number of matriculates was 421, and the number of graduates in March, 1830, was 127.

In 1865 the school still remained on Ninth Street. The number of chairs was still seven, the number of matriculates for the year was 520, and the number of graduates at the end of the session was 164.

The number of matriculates for the session of 1874–75, after the removal to this side of the river, was 371, and the graduates at the end of this session were 100 in number.

New positions and new names now appear in the reports of the medical school and the hospital—clinical professors of surgery, of medicine, of diseases of women and children, of the eye, of the ear, clinical lecturers on nervous diseases and on skin diseases, and a lecturer on pathological anatomy and histology. In addition to this list of professors and lecturers, didactic and clinical, a number of demonstrators, assistant demonstrators, assistants to the professors, etc., appear in the catalogue.

In 1904–05 the year when the new laboratories building was opened, the number of full professors had increased to twenty-one, with an attending host of professors of less rank, lecturers, demonstrators, associates, instructors, assistant instructors, and assistant demonstrators.

The last year, 1908–09, showed 559 students in attendance, 22 full professors, and clinical professors, associate professors, adjunct professors, and other teachers to the number of no less than 134. In a general way, these positions are the same as in 1904, but with a few additions and changes of title.

The number of those engaged in medical instruction at the University of Pennsylvania may seem large, especially to those trained a quarter of a century or more since. Foreign schools, however, and especially the French, have a much larger number of instructors of all grades. Dr. Osler, in his *Impressions of Paris*, which will be referred to more fully later in the address, tells us that in the University of Paris there are 37 professors, 43 agrégés or assistant professors, with scores of assistants and demonstrators in the different laboratories.

In many of the introductory lectures to which in either a general or special way I have called your attention in these remarks, the form of instruction which students on entering the University might expect to receive was more or less considered by the orator of the day. In some instances, as in one of Gibson's lectures, the student's attention was invited to a summary of the good things which each of the professorial incumbents had in store for him. In this way, I suppose, it was hoped that his appetite would be whetted for the feast of learning of which he was about to partake. I shall not venture to analyze for you the scope and character of the work with which you will all soon become familiar. I have thought, however, it might be of interest briefly to discuss undergraduate medical instruction in other countries, to the end that you might for yourselves compare it with our own curriculum.

Foreign medical schools have exerted a great influence on the development of the University of Pennsylvania, more especially in certain periods. The founders of the school were trained in the universities of Edinburgh and of Leyden, and some of them got at least a portion of their undergraduate experience in Padua and Paris, and brought with them to America ideas and plans gained at these great medical centres. Some were inspired for their work by the teaching of John and William Hunter, in London, or, at a later date, by the great surgeon Sir Astley Cooper. Stillé and others were attracted to Paris by the reputation of Louis and Laennec. In the earlier years, in most cases, these men of great aspirations went as undergraduates to foreign seats of learning, although this was not always the case, especially after the first two or three decades of the last century.

The home degree was sometimes taken before and sometimes after that which was given abroad, but both were usually secured by the ambitious student. Dr. Joseph Carson, who became professor of materia medica and pharmacy in the University in 1850, spent a considerable period in India as well as in Europe.

When specialism and laboratory work began to assert themselves abroad (1865 to 1875), some sought out the apostles of the new era—the specialists in ophthalmology, otology, dermatology, neurology, etc.—and came back from their pilgrimages to initiate the new departure in their own country. These men were usually postgraduate students in the foreign centres, and since then down to the present time, Americans studying medicine abroad have usually been students of this class.

How much they have profited will become known to you during your attendance upon some of the courses of instruction which have been prepared for you under the present roster.

It would not, however, be just to the history of our school and those who have honored it to lead you to believe that the medical education, undergraduate and postgraduate, afforded by European institutions and teachers has always been necessary to make good American doctors and teachers of medicine. Some of the greatest men who have honored the chairs of the medical department of the University of Pennsylvania, or have become illustrious as practitioners of medicine or surgery, have never had the advantages of foreign training. To illustrate this truth I have only to recall to you the names of such men as Agnew, Leidy, Goodell, Ashhurst, and Horatio C. Wood, and many others of our own and earlier days.

One of the chief reasons perhaps why undergraduates do not consider it worth while to receive their degrees abroad is to be found in the fact that the instruction at home is so much improved as to make this not only unnecessary but undesirable. Of course, I mean the instruction which is given in the best of our American schools, like the one which you honor with your presence this evening.

I doubt not that the day will come when both the undergraduate and the postgraduate students of Great Britain, Germany, Austria, France, Italy, and other nations of Europe and the Orient will seek our shores because of the celebrity of our schools. When this time comes we can in some degree repay the debt of gratitude which this and other American schools have owed to other nations from the days of Boerhaave, Cullen, and the Hunters.

During your next four years of attendance here you will doubtless hear much of improvements which have been made and those which are contemplated in the immediate or more remote future. You will form your own ideas as to the value of present methods, these impressions taking color not only from your personal observation, but from your inclinations and mental characteristics. You will, I trust, see much to approve; doubtless also much which you will think could be better done. In both instances you will be correct, but when your minds take a critical turn, you should think both of the history of the institution and also of the present state of medical teaching in other schools in this country and abroad. Probably, after all, no school has so well kept pace with progress as that in which you have become students. Probably no school offers greater laboratory and clinical opportunities—the latter in particular, because not only of the hospital connected with the school, but also because of the proximity of one of the greatest of city hospitals to its campus, are not surpassed by those of any other American institution. When you reach your third and fourth years you will begin to appreciate these opportunities.

Let us now consider the methods of undergraduate medical instruction in France, Germany, and Great Britain.

Let me first direct attention to the French schools. A few months since, in the *Journal of the American Medical Association* (Vol. lii, Nos. 9, 10, and 11, February 27, March 6 and 13, 1909), Dr. William Osler, once an honored clinical professor in this University, and now Regius Professor of Medicine in Oxford University, England, and Honorary Professor of Medicine in Johns Hopkins University, under the title, *Impressions of Paris*, published some interesting experiences and reflections on the medical schools and teachers of the French metropolis. From these impressions, and also with the kind assistance of a graduate of the University of Paris, I am able to present some facts regarding the methods of undergraduate medical instruction in France.

The requirements for admission to a course in medicine in France are a diploma from a lycée or college, the course in which corresponds in large part with that given at a first-class English or American college or a German gymnasium. These requirements are, therefore, somewhat in advance of our own, but we are rapidly reaching up to them, as those of the entering class know. It may not be out of place here to quote from the University of Pennsylvania

Catalogue, Fasciculus of the Department of Medicine (1908-1909), the announcements of the entrance requirements for 1909–1910 and 1910–1911.

For the academic year 1909–1910, "Candidates must have successfully completed work equivalent to that prescribed for the Freshman Class in colleges recognized by this University, which must include a knowledge of Physics, Chemistry, and General Biology or Zoölogy, together with appropriate laboratory work in each of these subjects, as specified by the College Entrance Examination Board, and two foreign languages, one of which must be French or German. . . . Certificates from recognized colleges covering these requirements will be accepted in lieu of an examination."

For the academic year 1910–1911, "Candidates must have successfully completed work equivalent to that prescribed for the Freshman and Sophomore Classes in colleges recognized by this University, which must include a knowledge of Physics, Chemistry, and General Biology or Zoölogy, together with appropriate laboratory work in each of these subjects, as specified by the College Entrance Examination Board, and two foreign languages, one of which must be French or German. . . . Certificates from recognized colleges covering these requirements will be accepted in lieu of an examination.

"Exceptions to above. Candidates who have had insufficient preparation in Physics, Chemistry, General Biology or Zoölogy, but who have successfully completed at least three years of an accepted college course, will be admitted with conditions in these subjects."

The undergraduate French student must attend regularly and systematically at a given school, as the University of Paris, for at least five years. He passes examinations each year, or if he prefers he can be examined after the first year every six months. The student is assigned to do work at a certain hospital, and the chief of the service of the hospital certifies to the fact that he has been in regular attendance. With regard to hospitals, both the duties and privileges of the entering student in France differ greatly from those belonging to the student in this country. From the first he is expected to attend the hospitals, not, as with us, to postpone such attendance until his third or fourth year. Some difference of opinion exists even in France as to the value of this method, but it is firmly adhered to and in general highly regarded by those who are immediately concerned with the teaching of medicine. Its defenders believe that the student should from the very first get more or less acquaintance with those suffering from diseases which he will be called upon to recognize and treat in his life's work. Others hold with Dr. Osler that it would be better for the student to spend his first year or two years in obtaining a thorough grounding in the sciences fundamental to practical medicine.

Besides his clinical work, the French student has laboratory work, dissection, histology, pathology, bacteriology, etc., regarding which also he must have certificates of attendance and work. He can go up for final examinations at the end of five years, or continue for an indefinite period. The final examinations are usually given on subjects in which the students have not already passed. Formal questions of principle or theory are asked in the first year. The next year in the examination on pathological anatomy gross specimens are shown, and the student is required to construct a picture of the disease represented by the specimen. The clinical examinations consist in the presentation of patients, usually three, and the student is given about fifteen minutes to make a diagnosis. After the student has passed all the examinations, a thesis must be presented and printed at his expense. At the very last he comes up before a committee of at least four of the Faculty and defends his thesis in public.

A limited number of students can by competitive examination associate themselves with certain physicians and surgeons of the hospitals under the title of externes or internes, but a man cannot become an interne until he has passed at least three years as an externe. The externe corresponds in considerable degree to the interne or resident physician of a hospital in this country. The higher status of the student externe in France as compared with the student in this country is worthy of note. At one time in the history of the Philadelphia Hospital students held a somewhat similar position to that of the French externe. They were what were termed "resident pupils," but now and for many years in all American hospitals the resident physicians or internes are necessarily graduates of some medical school, and in addition have usually passed the examination of the State board.

The position of interne in a French hospital is one eagerly sought and of much importance. Many apply for it, and but few are chosen. The positions are filled, as in the case of externes, by competitive examinations under the custom of the concours. The concours or public examinations play a great role in the history not only of the student, but of the aspiring physician.

"The interne," says Osler, "is a special French product, unlike anything else in the medical world. He is still a student, yet he has all the responsibility of a practitioner, and he is house surgeon, house physician, clinical assistant, laboratory assistant, special research student rolled into one. He lives in the hospitals for four years, a sufficient length of time to give him an exceptionally good education and a large experience. He comes into delightful relations with his chiefs, he lives in charming comradeship with his fellows, and if there is anything good in him he finishes his term with an admirable bit of original work which appears as his thesis for the M.D. He cannot take his degree while an interne."

Various positions connected with the universities and with hospitals in Paris and throughout France can be competed for by those who have served as internes. These positions must be held in succession from the lowest to the highest. No position is obtained through recommendation or without a competitive examination.

As this address is not concerned with postgraduate work, I shall not discuss with you, although it would be an interesting topic, the manner in which the concours or public examiations are held for the agrégés, which correspond somewhat to our associate or assistant professors. Only those who have been internes while students can compete for these positions.

The manner of dedication of the thesis by the French medical graduate is worthy of passing note. With our American lack of sentiment and reverence, or, rather, I should say, our custom of not so frequently expressing the sentiment and reverence in print, these dedications seem peculiar and perhaps to some amusing. The dedication of the thesis in most instances extends over two or more pages. In one at which I glanced, not less than six pages were devoted to the expression of the graduate's affection and homage. In the first place, it seems a universal custom, and a pleasant one, for all the living members of the graduate's immediate family to be recognized, and even his collateral relatives and friends are not forgotten. The president of the thesis (he who presides when the thesis is defended by the student) is universally included in the elaborate dedication, and not infrequently members of the Faculty, of the hospital staffs, and others concerned in his medical education. His father, mother, brothers, sisters, grandfather, grandmother, aunts, cousins and nieces, brothers-in-law and sisters-in-law, and even at times the mother-in-law and stepmother come in for a share of the homage. After all, it is a charming custom, even if it may seem to us, in our unconventionality, at times a little overdone.

The system of medical education in Germany is different from that of any other country. That it has much to recommend it there can be no doubt, but whether, all things considered, it is the best system, may be open to discussion. Recently I said to a young German ship surgeon, "Where did you receive your medical training?" He replied, "Breslau, Kiel, Heidelberg, Leipzig, Berlin," etc., naming in all at least some five or six well-known German universities. If a young American doctor, in answer to a similar inquiry, should tell his interlocutor that his medical education had been received at the University of Pennsylvania, Jefferson, Tulane, the College of Physicians and Surgeons, Chicago, and Ann Arbor, it is possible he might create the impression that he had become persona non grata at some of the institutions named.

A German course in medicine is often, in truth, a pilgrimage. The young man, after acquiring the proper credentials by graduation at the Gymnasium, decides for some reason that he will take his first semester at some one of the numerous German universities. His reason for the choice is not always the same; it may be the celebrity of the teachers; it may be because his forebears were educated partly at this institution; it may be because of some corps which he desires to enter, or because of the pleasant student life of the university town. Whatever the reasons, and they are diverse, his name is inscribed in a book which he afterward carries with him until the time of his graduation, wherever he may graduate. He finishes his first year, or it may be only his first semester, is properly certified to this effect by inscription in his book and by other methods, and passes for another semester or another year to another university. He may thus continue, so that before the time of his graduation it is possible for him to have attended as many as seven or eight universities, although this large number is not perhaps the rule.

For the German student there is no roll-call, no registration of attendance, no close supervision by dean or professor; he has that which has become so dear to all of his class, academic freedom. It is not to be presumed, however, that he gets through his course without giving some evidence of work and ability. The test comes at the time of examination. At the end of two years, at whichever university he happens to be, he must appear for the "Tentamen Physicum" examination in the fundamental subjects, and at the end of his last year he must make a good showing by examinations of his knowledge in the more practical branches of medicine and surgery. If in his evening dress coat, white tie, high hat, and kid gloves, he is able to withstand the searching investigation of the Herren Professoren as to his acquirements, he is finally by proper process certified for the reception of his degree. He is also required later, before he can practise, to pass the state examination. It is, indeed, not necessary that he should receive the degree, but there is one difference between one who receives the degree and passes the state examinations, and one who only is successful at the latter: The former forever after has the right to be addressed as Herr Doctor; the latter, however well qualified he may be, is spoken of only as Herr Arzt. I am not altogether satisfied that the German method would flourish on American soil, but will leave the matter for your consideration.

I have heard some curious reasons given for the German student's preferring this or that university. Jena, for instance, is popular with many partly because of its medieval customs, the free rein which the student has for his gentle follies, and the extraordinary manner in which his beer is supplied. A gentleman who recently visited Jena is my authority for the statement that only one innkeeper in that town ever demands immediate payment from the student for the beer which he drinks. When a new student lands in the other cafés of Jena he is cordially invited by mine host to drink of his beer without stint and not bother about paying for it. He is expected, however, to keep a charge account which he may liquidate at some future date. The proprietor (recently deceased), jocularly called "Kammer Karl" by the students, of a favorite "Bier Lokal" of Jena is said to have had an account of two hundred thousand marks standing against the students and former students of the university, and this apparently without causing him any worriment. Sometimes the student pays next month, or next year, or in the years to come when he has become a prosperous physician. As time goes on one year compensates for another, and the dispenser of beer ever remains satisfied.

Heidelberg is said to be a favorite university for attendance in the summer, because of its charms of various sort, including the great tun, and the many opportunities for duelling and other like amenities.

In the schools of Great Britain the requirement for admission to a medical course is: If the student has not received the degree of B.A., he is obliged to pass the matriculation examination of the college or school in which he intends to pursue his studies. He can, if he desires like the German student, pass from one hospital school to another from year to year, but, as a rule, he does not do this, sticking pretty closely to the school he enters. His attendance at the school must be five years, during which time he is obliged to complete yearly graded courses, passing the required examinations.

After graduation from a hospital or university school other examinations are required before the right to practise is obtained. Several grades as to standing and privileges are recognized. In the first place, the physician or surgeon by examination can become a licentiate of the Society of Apothecaries (L.S.A.), which entitles him to become what would be called in America a general practitioner. He has the right to practise both medicine and surgery, and can sue for his bills, whereas if a fellow of the Royal College of Physicians is not thus licensed he cannot, by a rule of the college, sue for his bills. The term apothecary is used in a different sense from that in which it is employed in this country. Here it means a compounder of drugs, while in Great Britain it means one who is entitled to practise medicine and surgery.

Besides the licentiates of the Society of Apothecaries, two other grades are recognized, those of members and Fellows of the Royal Colleges of Physicians and of Surgeons. To become a member of the Royal College of Physicians or of the Royal College of Surgeons (M.R.C.P.) and (M.R.C.S.), a different and more severe examination is required than that for licentiate of the society of apothecaries. To become a fellow of the Royal College of Physicians, or of the Royal College of Surgeons (F.R.C.P.) and (F.R.C.S.) still other and more exacting examinations are required.

A number of the British universities, as those at Oxford, Cambridge, Edinburgh, Manchester, Liverpool, Dublin, etc., have medical schools connected with them. At these the students, after a supervised course of five or six years, on passing their examinations successfully, during, and at the close of the course, can receive the degree of bachelor of medicine. The degree of doctor of medicine is given not less than one year later on the presentation of a thesis, which must be accepted by the university board. Licentiates of the Society of Apothecaries, and members, or even Fellows, of the Royal Societies of Physicians and of Surgeons, are not entitled to write the degree of M.D. after their names or the prefix Dr., unless they have obtained the degree at some university or college in regular course. Many doctors go to Brussels or to one of the provincial medical colleges as to that of Liverpool or Manchester to receive the degree of M.D., so as to make use of this title.

In Canada, at the Toronto University, and in the McGill University in Montreal, the requirements for admission and the course of instruction are practically the same as in England. Graded examinations are passed during the courses. At the end of the second year a primary examination before the medical council of the province can be, and usually is, taken, although this is sometimes postponed until the completion of the full course of attendance, when all the examinations are taken at the same time.

Some of the Canadian universities give M.B., and later M.D., for the latter requiring a thesis. Others grant the M.D. at the end of the four or five year course. The length of the course in Canada averages about five years. A few years since it was four years, and now a movement is on foot in Toronto to make it six years.

In the English Hospital schools and in the medical department of the English universities the supervision of the student is systematic and in most instances thorough.

The opportunities for hospital work are considerable in the great cities like London. So far as I know few opportunities are offered to the English student to obtain positions by competition except those of interne and registrar.

I have called attention to the forms of instruction of the medical student abroad, not simply for your entertainment and information, but to incline you, during your college life, to compare these educational schemes with our own. In requirements for admission Germany, France, and England are still somewhat in advance of us, but this will be very little, if any, when our recently adopted requirements are in full action. In the matter of supervision and competition we could probably copy something from the French, in that of academic freedom, something from the Germans, especially as regards the last year or the last two years of the medical course. This should be not less than five years, a conclusion to which all thoughtful medical educators and students are coming. The fifth year, however, should be largely an elective one, although the degree could not be given until the end of this year, unless, indeed, we go back to our own original method and that which prevails to some extent at the present time in England, of first giving the degree of bachelor of medicine and after another year that of doctor of medicine.

In conclusion, let me say that good doctors and medical teachers, and even great ones, have been produced by every method of medical instruction in vogue since and before the days of Stahl and Boerhaave. After all, the young man of good mentality and the right metal will find his way to the achievement of his ends under any method, although it is the duty of a school to make this the best possible.

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THE CLINICAL PICTURE OF MULTIPLE SCLEROSIS WITH THE PATHOLOGICAL FINDINGS OF ARTERIOSCLEROSIS

By Charles K. Mills, M.D., and William G. Spiller, M.D.

One of the speakers (Dr. Spiller) showed some years ago in connection with Dr. Camp that a mistaken clinical diagnosis may easily be made regarding multiple sclerosis, as the symptoms in a case of cerebrospinal syphilis were sufficiently like those of multiple sclerosis to justify the diagnosis of the latter disorder. More recently he has demonstrated with Dr. Woods, what has also been proven by others, that areas of sclerosis produced by syphilis may exist without any symptoms sufficient to lead to a diagnosis of multiple sclerosis. Dr. Mills and he had recently had another case in which the symptoms of this latter disease were so distinct that the diagnosis of multiple sclerosis seemed assured until a microscopical examination revealed the error. There are few diseases in which errors of diagnosis are more likely to occur, and therefore clinical data deduced from purely clinical examinations are likely to canse mistakes, if too sweeping conclusions are drawn from them. The authors cautioned therefore against wide-reaching conclusions relating to multiple sclerosis when necropsies are wanting.

The case reported is as follows:

Bridget D., was admitted to the Philadelphia General Hospital, Dec. 19, 1892. The history is somewhat uncertain, but it is probable that the symptoms began at some period before her admission to the hospital. She had done hard work, such as washing. She was a white woman, past middle life. She had coarse tremor of the face, arms and hands. The tremor of the head developed later, as well as the halting speech. The tremor of the head was a combination of vertical and rotary movement. Speech required considerable effort, and there was hesitation before the utterance of each word. Festination was not present, although the patient had a stooping posture. The patellar reflex of the right extremity was normal, but that of the left was almost lost. Romberg's sign was not present. There was no appreciable loss of voluntary power or of sensation. When the patient stood the entire trunk was in tremor, the upper and lower limbs vibrating. There was a coarse intention tremor of the hands and head, in the latter a lateral as well as an up and down movement. The station was worse by closing the eyes. The tremor was less when the patient was lying down, but did not entirely cease.

A note made in 1907 states that the patient had shown signs of mental failure, she had an abnormal appetite, and frequently stole her neighbor's meals, at other times she secreted other patients' clothing beneath her bed, until she had five or six dresses concealed there. She answered questions correctly.

She died of pneumonia October 28, 1907.

The important symptoms in this case were intention tremor and scanning speech, each of extreme type. The tremor was so intense that when she attempted to put the finger to the nose most violent intention movements would occur, and standing would cause an intention tremor of the whole body. The speech was typically scanning and somewhat explosive, the words were jerked out. The patient was very irritable, and avoided examination. She manifested no other symptom of multiple sclerosis, but the scanning speech and intention tremor were so indicative of multiple sclerosis that the clinical diagnosis of this disease was never questioned by anyone who saw the patient.

A microscopical examination of the brain and cord gives the following results: The vessels of the spinal cord, especially those of the cervical region, show pronounced arteriosclerosis. Some round cell infiltration is found in the pia about the vessels in the medulla oblongata, but this is slight even here, and is insignificant in the spinal cord. The nerve cells of the anterior horns are intensely pigmented in the cervical and lumbar regions. Many amyloid bodies are found in the spinal cord. The condition is such as is observed in certain cases of paralysis agitans or senility, and in no way resembles that seen in multiple sclerosis.

Dr. J. Hendrie Lloyd said he had felt very much interested in the subject of senile tremor for a long time. He does not think we have gotten to the bottom of the subject. The case narrated seemed to him to have been one of rather aggravated so-called senile tremor. She had, it is true, some hesitation of speech, but no nystagmus, no exaggerated knee-jerks. It is not difficult to distinguish such a case from multiple sclerosis. The more difficult thing is to tell just what causes it. Senile tremor sometimes differs very decidedly from paralysis agitans, at other times it resembles it. We have a head tremor more marked and sometimes the slight impairments of speech. When we consider multiple sclerosis, occurring preferably in young women, with nystagmus and exaggerated knee-jerks, and with symptoms of lateral sclerosis more or less marked in most cases, it does not seem that the differentiation of it from the senile type is so difficult. A woman was in Blockley with symptoms at first consisting of almost primary lateral sclerosis. She is now in bed, for she cannot walk. She has highly exaggerated knee-jerks, with a spastic condition of the lower extremities. She has a slight intention tremor, a little suspicion of nystagmoid movements of the eyeballs, and a little slurring speech. This is one of those cases in which diagnosis is at first difficult. She is 36 or 37 years of age, a little beyond the period in which multiple sclerosis develops as a rule.

That you can have syphilitic disease simulating multiple sclerosis is an undisputed fact. Dr. Lloyd believed the statement had been made by some that in disseminated sclerosis there is no lymphocytosis of the cerebro-spinal fluid, and that this may be used as a means of differentiation from spinal syphilis. Recently Raymond reported a case which seemed to be rather typical of disseminated cerebral sclerosis in which he found marked lymphocytosis; so the point does not seem definitely settled.

Dr. D. J. McCarthy said that he had put on record with Dr. Charles W. Burr, a case at the Home for Incurables of disseminated syphilis giving a typical clinical picture of the classic form of disseminated sclerosis. They also recorded a case with no intention tremor, no scanning speech, no nystagmus, only mental defect. In other words unless you have in disseminated disease throughout the cerebro-spinal axis a certain distribution of it, you will not get these symptoms of multiple sclerosis. Exactly what the position of these foci should be has not been determined, probably the pons and medulla so far as the nystagmus and scanning speech are concerned. Dr. McCarthy agreed with Dr. Lloyd that the thing that leads him to a diagnosis is not so much intention tremor, as the fact that these symptoms are associated with a spastic condition of the lower extremities. There are a sufficient number of cases on record in which this spasticity has not been present, in which the condition of atrophy has been presented with a condition of disseminated sclerosis.

Dr. Spiller said that the difficulty in diagnosing multiple sclerosis from other diseases depends on the diffuseness of the symptoms. Multiple sclerosis is a disease which may affect all parts of the central nervous system. Therefore we can readily understand that we may get similar symptoms from syphilis in certain instances. The charge has been made that we in this country do not know how to diagnose multiple sclerosis, that we wait for typical cases such as Dr. Lloyd speaks of, which anyone can diagnose. Multiple sclerosis is by no means always accompanied by spasticity. The patient reported by Dr. Mills and himself had entered the hospital seventeen years ago, and the tremor was not senile. It is well known that Charcot denied the existence of senile tremor.

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